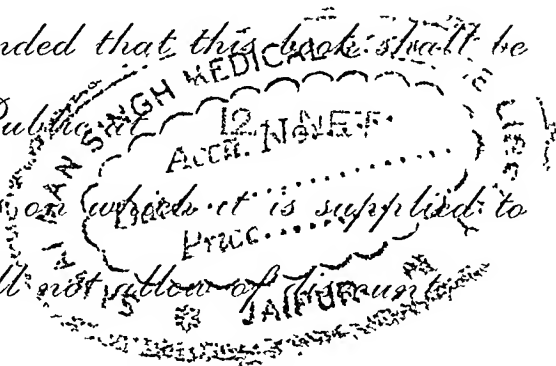


Livingstones' Medical Handbooks.

Clarkson & Farquharson's Student's Handbook of Physiology	net 12/-
M'Lachlan's Applied Anatomy: Surgical, Medical, and Operative, 2 vols., revised by A. A. Scot Skirving (4th Edition).....	" 18/-
Glaister's Medical Jurisprudence, Toxicology and Public Health, <i>Illustrated</i>	" 15/-
—— Manual of Hygiene (2nd Edition)	" 6/-
—— Poisoning by Arseniuretted Hydrogen	" 5/-
Mikulicz's Tubercular Joint Disease	" 4/6
Wheeler and Jack's Medicine and Therapeutics (3rd Edition) ..	" 8/-
Husband's Forensic Medicine, Toxicology and Public Health (7th Edition)	" 10/6
—— Practice of Medicine (6th Edition), Revised by R. F. C. Leith and R. A. Fleming.....	" 12/-
—— Examination of the Urine (4th Edition)	" 1/-
Examination Questions, Practice of Medicine, with Answers, Part 1—GENERAL DISEASES. Part 2—SKIN DISEASES, Part 3—FEVERS.....	each net 1/-
Craig's Posological Tables (New Edition).....	" 1/-
Masterman's Elementary Text-Book of Zoology (2nd Edition), <i>Illustrated</i>	" 12/-
Orr's Handbook of Public Health.....	" 4/-
Handbook of Skin Diseases, with <i>coloured illustrations</i>	" 2/6
Kempe's Diseases of the Eye.....	" 1/6
Wishart's Botanist's Vade Mecum	" 2/-
—— Schedules for Plant Description	" 6d.
Webster's Puberty and the Change of Life.....	" 1/-
Anatomy— <i>Catechism Series</i> , Six Parts—each	" 1/-
Bacteriology— Do.	" 1/-
Botany— Do. Two Parts—each ..	" 1/-
Chemistry— Do. Two Parts—each	" 1/-
Forensic Medicine— Do.	" 1/-
Histology— Do.	" 1/-
Materia Medica— Do. Three Parts—each	" 1/-
Midwifery— Do. Two Parts—each	" 1/-
Pathology— Do. Parts I., II. & III.—each.....	" 1/-
Physics— Do. Two Parts—each	" 1/-
Physiology— Do. Four Parts—each	" 1/-
Public Health— Do. Five Parts—each	" 1/-
Surgery— Do. Seven Parts—each	" 1/-
Surgical Anatomy } Do Two Parts—each.....	" 1/-
and Operations.— }	
Toxicology— Do.	" 1/-
Zoology— Do. Two Parts—each	" 1/-
Johnstone's Zoology Notes	" 3/6

❖ NOTICE. ❖

It is intended that this book shall be
sold to the Public on (which it is supplied to
and the terms on which it is supplied to
the Trade will not differ from those of
E. & S. Livingstone.



HUSBAND'S
PRACTICE OF MEDICINE.

HUSBAND'S PRACTICE OF MEDICINE

DESIGNED FOR THE USE OF STUDENTS AND
PRACTITIONERS

SIXTH EDITION, REWRITTEN AND ENLARGED

ROBERT F. C. LEITH, M.A.

M.Sc., M.B., C.M., F.R.C.P.Ed.

Professor of Pathology and Bacteriology, Birmingham, &c., &c.

AND

ROBERT A. FLEMING, M.A.

M.D., F.R.C.P.Ed.

*Lecturer on the Principles and Practice of Medicine, School of Medicine
of the Royal Colleges, Edinburgh; Assistant Physician,
Royal Infirmary, Edinburgh,*

EDINBURGH

E. & S. LIVINGSTONE, 15 TEVIOT PLACE

1908

PRINTED BY
LIVINGSTONE & LOGAN
25 THISTLE STREET, AND 9 CALTON ROAD
EDINBURGH

PREFACE.

THIS edition of Husband's Practice of Medicine was begun some time ago. An attempt was made at first to carry out the wishes of the publishers for a revision of the book, which would bring it up to date, but afterwards a rewriting rather than a revision was found to be necessary. The aim and scope of the book, however, has been preserved, though its substance has been completely altered. It is hoped that it will continue to provide students with a concise, reliable and modern textbook of medicine. In a book of this size the difficulty is one rather of elision than of inclusion. The publishers decided to follow the practice adopted in former editions of leaving out illustrations. No separate heading has been given to diseases of the spleen or lymphatic glands. This step was taken, perhaps unwisely, chiefly because the changes in these organs are only a part of other diseases. It will be found that the elision is one more of form than of fact, for the diseases have been described under other headings, such as blood and blood-forming glands, and a reference to the Index will enable the student to acquaint himself with the diseases of these organs as well as if they had been described under separate headings. Much care and labour has been devoted to the Index, under the belief that an index never can be too full and that extra space given to it will more than compensate for elisions elsewhere. It is very complete. Not only does it afford exhaustive reference to the substance of the book but it will also enable the student to examine himself upon his knowledge.

Treatment throughout and the diseases of the nerves have been written by Dr Fleming, the rest of the book by myself.

R. F. C. LEITH.

CONTENTS



Section 1.

GENERAL PATHOLOGY.

I.—CIRCULATORY DISTURBANCES.

Hyperæmia,	3
Dropsy,	4
Thrombosis,	7
Embolism,	10
Hæmorrhage,	12
Inflammation,	14

II.—NUTRITIONAL DISTURBANCES.

Necrosis,	20
Atrophy,	22
Degeneration (Cloudy Swelling),	23
Fatty Degeneration,	24
Mucoid Degeneration,	27
Colloid Degeneration,	28
Hyaline Degeneration,	29
Lardaceous Degeneration,	29
Fatty Infiltration,	31
Calcareous Infiltration,	33
Pigmentation,	33
Hypertrophy,	36
New Growths,	37

Section 2.

GENERAL DISEASES.

I.—DISEASES DEPENDENT ON SPECIFIC POISONS.

THE INFECTIOUS DISEASES.

Pyrexia—Fever,	41
-----------------------	----

SUB-GROUP (α)—ACUTE INFECTIVE DISEASES OF
KNOWN BACTERIOLOGY.

Relapsing Fever,	52
Typhoid and Typhus,	57
Typhoid Fever,	58
Infantile Remittent Fever,	73
Epidemic Cerebro-Spinal Meningitis,	77
Epidemic Cholera,	80
Malta Fever,	87
Influenza,	89
Diphtheria,	92
Laryngeal Diphtheria,	102
Tetanus,	103

SUB-GROUP (β)—CHRONIC INFECTIVE DISEASES OF
KNOWN BACTERIOLOGY.

Tuberculosis,	109
Leprosy,	123
Actinomycosis,	128
Mycetoma or Madura Foot,	132

SUB-GROUP (γ)—SEPTIC DISEASES OF KNOWN
BACTERIOLOGY.

Sapraemia, Septicaemia and Pyaemia,	133
Erysipelas,	139
Puerperal Fever,	143

SUB-GROUP (δ)—INFECTIVE DISEASES OF UNCERTAIN BACTERIOLOGY.

1. *Not Endemic*—

Febricula,	144
Variola—Small-pox,	149
Varioloid,	157
Vaccinia—Cow-pox,	159
Varicella—Chicken-pox,	162
Scarlatina—Scarlet Fever,	163
Morbilli, Rubeola, or Measles,	172
Rötheln, Rubella, or German Measles,	175
Whooping-Cough,	178
Parotitis,	181
Syphilis,	182

2. *Endemic*—

Typhus Fever,	192
Yellow Fever,	201
Dengue,	205
Dysentery,	207
Beri-Beri,	211

SUB-GROUP (ε)—INFECTIVE DISEASES COMMUNICABLE FROM ANIMALS TO MAN.

Plague,	214
Glanders and Farcy,	219
Anthrax, or Splenic Fever,	222
Rabies—Hydrophobia,	225
Foot-and-Mouth Disease,	234

SUB-GROUP (θ)—DISEASES DUE TO PROTOZOA.

Malarial Fever,	236
Hæmoglobinuric Fever,	246
Psorospermiasis,	248
Sleeping Sickness,	250
Amœbic Dysentery,	207

II.—DISEASES CAUSED BY WORMS.

The Trematodes—Distomiasis,	254
The Cestodes—Tape-Worms—Hydatids,	255
Tænia Echinococcus—Hydatids,	258

CONTENTS.

The Nematodes—Round and Thread Worms, ...	261
Ascaris Lumbricoides—The Round Worm—	
Ascariasis,	261
Oxyuris Vermicularis—The Thread Worm, ...	262
Tricocephalus Dispar—The Whip Worm, ...	263
Anchylostoma or Dochmius Duodenale—Anchy-	
lostomiasis,	264
Trichina Spiralis—Trichiniasis,	266
Dracunculus Medinensis—The Guinea-Worm—	
Dracontiasis,	268
Filariasis,	269

III.—THE ARTHROPODA,	272
-----------------------------	-----

IV.—THE INTOXICATIONS.

Alcoholism,	274
Food Poisoning—Ptomaine Poisoning, ...	279
Snake Poison,	281
Opium Poisoning—Morphia—Morphinism ...	283
Cocaine Poisoning—Cocainism,	285
Tobacco Poisoning,	286
Metallic Poisoning,	287
Phosphorus—Phosphorism,	287
Arsenic Poisoning,	288
Lead Poisoning—Plumbism,	289
Sunstroke,	292

V.—GENERAL DISEASES OF OBSCURE ORIGIN.

Rheumatic Fever—Acute Rheumatism, ...	293
Chronic Articular Rheumatism,	301
Muscular Rheumatism,	303
Gonorrhœal Rheumatism,	304
Arthritis Deformans or Rheumatoid Arthritis, ...	307
Rickets,	313
Gout,	318
Diabetes Mellitus,	327
Diabetes Insipidus,	338

Section 3.

*DISEASES OF THE BLOOD, BLOOD-FORMING AND
DUCTLESS GLANDS.*

I.—DISEASES OF THE BLOOD.

Introduction,	340
---------------	-----	-----	-----	-----	-----

SUB-GROUP (α)—THE PRIMARY ANÆMIAS.

Chlorosis,	349
Pernicious Anæmia,	352

SUB-GROUP (β)—THE SECONDARY ANÆMIAS,	...	358
--	-----	-----

II. DISEASES AFFECTING THE BLOOD-FORMING GLANDS.

SUB-GROUP (α)—DISEASES AFFECTING MAINLY THE
WHITE CELLS.

Leukæmia,	360
Splenic Anæmia,	370
Chloroma,	372
Hodgkin's Disease,	374

SUB-GROUP (β)—THE HÆMORRHAGIC DISEASES.

Hæmophilia,	379
Purpura,	381
Scurvy,	384
Infantile Scurvy,	386

SUB-GROUP (γ)—SPECIAL CONDITIONS.

Burns,	387
Poisons,	388
Paroxysmal Hæmoglobinuria,	388

III.—DISEASES OF THE DUCTLESS GLANDS.

The Ductless Glands,	389
----------------------	-----	-----	-----	-----

SUB-GROUP (α)—THYROID GLAND,	390
Goitre,	392
Myxædema,	393
Cretinism,	395
Exophthalmic Goitre,	396
Tumors of the Thyroid,	400
SUB-GROUP (β)—THE SUPRARENAL OR ADRENAL GLAND.			
Diseases of the Suprarenal Body, .	..		401
Addison's Disease,	402
Tumors of the Suprarenals,	406
SUB-GROUP (γ)—THE THYMUS GLAND.			
Diseases of the Thymus Gland,		406

Section 4.

DISEASES OF THE CIRCULATORY SYSTEM.

I.—DISEASES OF THE HEART.

Malpositions,	408
Malformations,	408

SUB-GROUP (α)—DISEASES OF PERICARDIUM.

Hydropericardium,	410
Hæmopericardium,	410
Pneumopericardium,	410
Acute Pericarditis,	411
Chronic Pericarditis,	416

SUB-GROUP (β)—DISEASES OF MYOCARDIUM.

Parenchymatous Degeneration,	417
Fatty Degeneration,	417
Fatty Infiltration,	418
Waxy Degeneration,	419
Fibroid Degeneration,	419

Segmentation and Fragmentation of Myocardium,	420
Atrophy of Myocardium,	421
Myocarditis,	421
Tubercular Myocarditis,	423
Syphilitic Myocarditis,	423
Lesions of Coronary Arteries,	424
Hypertrophy of Heart,	425
Dilatation of Heart,	427
Aneurism of Heart,	429
Rupture of Heart,	430
Tumors of Myocardium,	431
Animal Parasites,	431

SUB-GROUP (γ)—DISEASES OF ENDOCARDIUM.

Acute Endocarditis,	432
Benign Endocarditis,	435
Malignant Endocarditis,	438
Chronic Endocarditis,	442
Mitral Incompetence,	445
Mitral Stenosis,	449
Aortic Incompetence,	451
Aortic Stenosis,	454
Tricuspid Regurgitation,	456
Tricuspid Stenosis,	456
Pulmonary Stenosis,	457
Pulmonary Incompetence,	457
Combined Valvular Lesions,	458

SUB-GROUP (δ)—FUNCTIONAL DISORDERS OF HEART.

Functional Heart Disease,	461
Palpitation,	463
Arrhythmia,	463
Tachycardia—Rapid Heart or Heart-Hurry,	464
Bradycardia—Slow Heart,	466
Angina Pectoris—Breast Pain,	467

II.—DISEASES OF THE BLOOD-VESSELS.

The Pulse,	471
-------------------	-----

SUB-GROUP (α)—DISEASES OF THE ARTERIES.

The Degenerations,	474
Inflammation—Arteritis,	475
Acute Arteritis,	476

Chronic Arteritis,	476
Atheroma,	478
Aneurism,	481
SUB-GROUP (β)—DISEASES OF THE VEINS, ...	487

Section 5.

DISEASES OF THE RESPIRATORY SYSTEM.

I.—DISEASES OF THE NOSE.

Acute Nasal Catarrh,	488
Chronic Nasal Catarrh,	490
Hay Fever,	491
Epistaxis,	493
Adenoids,	495

II.—DISEASES OF THE LARYNX.

Acute Catarrhal Laryngitis,	496
Cedema Glottidis,	497
Chronic Laryngitis,	498
Tubercular Laryngitis,	500
Syphilitic Laryngitis,	500
Tumors of the Larynx,	501
Spasmodic Laryngitis,	502

III.—DISEASES OF THE BRONCHI.

Acute Bronchitis,	503
Chronic Bronchitis,	507
Plastic, or Fibrinous Bronchitis, ...	509
Bronchiectasis,	511
Asthma,	514
Obstruction of Trachea and Bronchi, ...	517

IV.—DISEASES OF THE LUNG.

Emphysema,	518
Collapse (Atelectasis,)	522

SUB-GROUP (a)—INTERFERENCES WITH THE CIRCULATION IN THE LUNGS.

Passive Congestion,	525
Œdema,	527
Hæmorrhage,	528

SUB-GROUP (β)—INFLAMMATIONS OF THE LUNG.

Introduction,	532
Acute Non-Specific Pneumonias,	534
Lobar Pneumonia,	534
Lobular Pneumonia,	543
Purulent Pneumonia and Abscess of Lung,				549
Gangrene of Lung,	552
Chronic Non-Specific Pneumonias,	554
Acute Specific Pneumonias,	558
Typhoid Pneumonia,	558
Influenzal Pneumonia,	559
Pulmonary Tuberculosis,	561
Acute Miliary Tuberculosis,	561
Acute Aerogenic or Bronchogenic Tubercle,				564
Chronic Specific Pneumonias,	570
Chronic Pulmonary Tuberculosis or Chronic Phthisis,	570
Syphilitic Pneumonia,	590
Actinomycotic Pneumonia,	591
Tumors of the Lung,	591
Parasites of the Lung,	592

V.—DISEASES OF THE PLEURA.

Hydrothorax,	594
Hæmothorax,	595
Chylothorax,	596
Pneumothorax,	596
Acute Pleurisy,	598
Chronic Pleurisy,	606
Tumors of the Pleura,	606
Parasites of the Pleura,	607

Section 6.

DISEASES OF THE DIGESTIVE SYSTEM.

I.—DISEASES OF THE MOUTH.

Simple Catarrhal Stomatitis,	608
Aphthous Stomatitis,	609
Parasitic Stomatitis or Thrush,	610
Ulcerative Stomatitis,	610
Gangrenous Stomatitis—Cancrum Oris—Noma,			612
Suppurative Stomatitis,	613
Chronic Specific Stomatitis,	613

II.—DISEASES OF THE SALIVARY GLANDS.

Altered Conditions of Salivary Secretion,	...	615
Inflammation of the Salivary Gland,	..	615
Tumors of the Salivary Glands,	...	616

III.—DISEASES OF THE PHARYNX AND TONSILS.

Acute Pharyngitis,	617
Chronic Pharyngitis,	618
Membranous Pharyngitis,	619
Ulceration of Pharynx,	619
Acute Tonsillitis,	619
Follicular Tonsillitis,	619
Suppurative Tonsillitis or Quinsy,	620
Chronic Tonsillitis,	622

IV.—DISEASES OF THE ŒSOPHAGUS.

SUB-GROUP (α)—ALTERATIONS OF THE LUMEN.

Diverticulum,	624
Stenosis,	625

SUB-GROUP (β)—INFLAMMATIONS OF THE ŒSOPHAGUS.

Acute Œsophagitis,	626
Chronic Œsophagitis,	626

SUB-GROUP (γ)—CANCER OF THE ŒSOPHAGUS.

Cancer of the Œsophagus,	627
--------------------------	-----	-----	-----

V.—DISEASES OF STOMACH.

Dyspepsia,	628
Table of Causes, &c., of Dyspepsia, ...	634
Degenerations of Stomach,	636
Malformations and Displacements,	636
Stenosis of Stomach,	637
Hour-Glass Deformity of Stomach,	637
Displacement of Stomach,	637
Inflammations,	638
Gastritis,	638
Simple Acute Gastric Catarrh,	638
Simple Subacute Gastric Catarrh,	640
Simple Chronic Gastric Catarrh,	641
Toxic Gastritis,	648
Mycotic Gastritis,	649
Acute Dilatation of Stomach,	650
Chronic Dilatation of Stomach,	650
Ulcer of the Stomach,	653
Duodenal Ulcer,	660
Cancer of Stomach,	661
Diagnostic Table of Gastric Cancer, &c.,	668

VI.—DISEASES OF THE INTESTINE.

Malformations and Displacements,	672
Intestinal Obstruction,	672
Degenerations of Intestines,	678
Circulatory Disturbances of the Intestines,	678
Inflammations,	679
Enteritis,	679
Colitis,	684
Psilosis, or Sprue,	684
Appendicitis,	687
Tuberculosis of Intestines,	694
Table of Intestinal Ulcers,	696
Syphilis of Intestines,	697
Tumors of Intestines,	698
Diarrhœa,	698
Constipation,	701
Intestinal Colic,	704
Diagnostic Table of Colic, &c.,	706

VII.—DISEASES OF THE LIVER.

Degenerations of the Liver,	707
------------------------------------	-----

Circulatory Disturbances,	707
Hyperemia,	707
Thrombosis of the Liver,	708
Inflammations,	709
Acute Hepatitis of the Liver,	709
Suppurative Hepatitis—Abscess,	710
Chronic Hepatitis—Cirrhosis,	713
Yellow Atrophy of the Liver,	724
Tumors of the Liver,	727
Hydatids of the Liver,	729

VIII.—DISEASES OF THE GALL BLADDER AND ITS DUCTS.

Gall Stones,	730
Cholecystitis,	737
Angiocholitis,	738
Icterus Neonatorum,	741
Suppurative Angiocholitis,	742
Cancer of Gall Bladder and Bile Ducts,	743
Obstruction of Bile Ducts,	745

IX.—DISEASES OF THE PANCREAS.

Hæmorrhage of the Pancreas,	748
Inflammation of the Pancreas,	749
Tumors of the Pancreas,	752
Cysts of the Pancreas,	753
Calculi of the Pancreas,	754

X.—DISEASES OF THE PERITONEUM.

Ascites,	757
Peritonitis,	757
Subphrenic Abscess,	765
Pelvic Peritonitis,	767
Chronic Peritonitis,	768
New Growths in the Peritoneum,	769
Cysts of the Peritoneum,	770

Section 7.

DISEASES OF THE KIDNEYS.

Malformations and Malpositions,	771
Degenerations—Waxy Degeneration,	773

CONTENTS.

xix

DISTURBANCES OF THE RENAL CIRCULATION.

Congestion,	777
Infarctions,	778

DISTURBANCES OF THE RENAL SECRETION.

Normal Constituents,	779
Amount of Urine—Anuria,	780
Amount of Urea—Uræmia,	782
Amount of Uric Acid,	784
The Urinary Salts,	785
Abnormal Constituents,	787
Albuminuria,	787
Hæmaturia,	790
Hæmoglobinuria,	791
Carbohydrates,	792
Pigments,	793
Tube Casts,	793
Calculi—Nephrolithiasis,	795

INFLAMMATION OF THE KIDNEY.

Nephritis,	798
Acute Nephritis—Acute Bright's Disease,	799
Chronic Nephritis—Chronic Bright's Disease,	806
Chronic Parenchymatous Nephritis,	806
Chronic Interstitial Nephritis,	809
Tuberculosis of the Kidney,	818
PYELITIS,	820
HYDRONEPHROSIS,	822
CYSTIC DISEASE OF THE KIDNEYS,	824
TUMORS OF THE KIDNEY,	826
PERINEPHRIC ABSCESS,	829

Section 8.

DISEASES OF THE NERVOUS SYSTEM.

Introduction,	831
The Motor System,	834
The Sensory System,	841

DISEASES OF THE MENINGES.

Dural Hæmorrhage,	844
Pachymeningitis,	845
Tumors of the Dura,	848
Leptomeningitis,	849
Acute Primary Leptomeningitis,	849
Tubercular Meningitis,	850
Meningococcal Meningitis,	853
Pneumococcal Meningitis,	853
Acute Secondary Leptomeningitis,	854
Chronic Leptomeningitis,	856
Tumors of the Pia Arachnoid,	856

DISEASES OF THE SUBSTANCE OF THE BRAIN AND CORD.

Degenerations of the Brain and Cord,	857
Hydrocephalus,	858
Hydromyelia and Syringomyelia,	862
Spina Bifida,	865
Circulatory Disturbances,	865

RUPTURE OF VESSELS.

Cerebral Hæmorrhage,	867
Spinal Hæmorrhage,	879
Hæmatorrhachis,	879
Hæmatomyelia,	881
Caisson Disease—Diver's Paralysis,	882
Embolism and Thrombosis,	884
Hemiplegia in Children,	886
Aneurisms of the Cerebral Arteries,	888
Thrombosis of the Cerebral Sinuses and Veins,	889

INFLAMMATION OF THE SUBSTANCE OF THE BRAIN (ENCEPHALITIS) AND SPINAL CORD (MYELITIS).

Introduction,	890
Acute Encephalitis,	891
Suppurative Encephalitis—Abscess,	893
Chronic Encephalitis,	896
Myelitis,	897
Acute Poliomyelitis or Infantile Paralysis, or Atrophic Spinal Paralysis,	904
Acute and Subacute Poliomyelitis in Adults,	910
Landry's Paralysis—Acute Ascending Paralysis,	910
Chronic Myelitis,	912

DISEASES OF THE WHOLE MOTOR SYSTEM—FIRST AND SECOND NEURONES.

Chronic Anterior Poliomyelitis—Progressive Muscular Atrophy,	913
Bulbar Paralysis—Glosso-labio laryngeal Paralysis,	917
Progressive Neural Muscular Atrophy—the Peroneal Type,	920
The Muscular Dystrophies,	921

DISEASES OF THE UPPER MOTOR NEURONE.

Infantile Spastic Paralysis—Spastic or Infantile Diplegia—Birth Palsies—Little's Disease,	924
Primary Spastic Paralysis of Adults—Primary Lateral Sclerosis—Primary Spastic Paraplegia,	926
Secondary Spastic Paralysis,	927

DISEASES OF THE LOWER MOTOR NEURONES, ... 928

DISEASES OF THE SYSTEM OF SENSORY NEURONES.

Tabes Dorsalis—Locomotor Ataxia—Posterior Spinal Sclerosis,	929
Herpes Zoster—Shingles—Zona,	938

COMBINED SYSTEM DISEASES.

Ataxic Paraplegia,	940
Friedreich's Ataxia—Hereditary Ataxia,	941
Cerebellar Hereditary Ataxia,	943
Anæmic and Toxic Combined Sclerosis,	943

SCATTERED NON-SYSTEM DISEASES.

General Paralysis of the Insane—Progressive General Paralysis—Dementia Paralytica,	944
Multiple Cerebro-Spinal Sclerosis—Disseminated Sclerosis—Insular Sclerosis—Sclerose en Plagues,	948
Tumors of the Brain and Cord,	951
Disorders of Speech,	957

GENERAL OR FUNCTIONAL DISEASES.

Paralysis Agitans—Parkinson's Disease—Shaking Palsy,	962
Acute Chorea—Sydenham's Chorea—St Vitus' Dance,	965

Chronic Chorea—Huntingdon's Chorea,	...	968
Senile Chorea,	...	969
Rhythmic or Hysterical Chorea,	...	969
Chorea Major,	...	969
Saltatory Spasm,	...	969
Habit Spasm,	...	970
Impulsive Tic (Gilles de la Tourette's Disease),	...	970
Epilepsy,	...	970
Eclampsia—Infantile Convulsions,	...	977
Occupation Neuroses—Professional Spasms,	...	978
Tetany,	...	980
Paromyoclonus Multiplex—Myoclonia,	...	982
Myasthenia Gravis,	...	982
Migraine—Hemicrania—Sick Headache,	...	983
Hysteria,	...	985
Neurasthenia,	...	991
Traumatic Neuroses,	...	994

VASOMOTOR AND TROPHIC DISEASES.

Angio-Neurotic Œdema,	...	995
Raynaud's Disease,	...	996
Erythromelalgia (Red Neuralgia),	...	999
Scleroderma,	...	999
Facial Hemiatrophy,	...	1000
Acromegaly,	...	1001
Hypertrophic Pulmonary Osteoarthropathy,	...	1002
Osteitis-Deformans—Paget's Disease,	...	1002
Osteomalacia—Mollities Ossium,	...	1003
Achondroplasia,	...	1003

DISEASES OF NERVES.

Neuralgia,	...	1004
Local Neuritis,	...	1012
Ascending Neuritis—Neuritis Migrans,	...	1014
Sympathetic Neuritis,	...	1014
Multiple Neuritis—Peripheral Neuritis,	...	1015
Tumors of Nerves,	...	1019

DISEASES OF SPINAL NERVES.

Paralysis of the Phrenic Nerve,	...	1020
Paralysis of the Brachial Plexus,	...	1022
The Lumbar Plexus,	...	1026
The Sacral Plexus,	...	1026

DISEASES OF CRANIAL NERVES.

First or Olfactory Nerve,	1028
Second or Optic Nerve,	1029
Optic Neuritis—Papillitis—Inflammation of the Optic Disc,	1031
Retro-Bulbar Neuritis,	1033
Optic Atrophy,	1033
Affections of the Retina,	1034
The Third Nerve,	1036
The Fourth Nerve,	1039
The Sixth Nerve,	1040
The Fifth or Trigeminal Nerve,	1043
The Seventh Nerve,	1048
The Eighth or Auditory Nerve,	1054
Meniere's Disease,	1056
The Ninth or Glosso-pharyngeal Nerve,	1058
The Tenth, Vagus or Pneumogastric Nerve,	1059
The Eleventh or Spinal Accessory Nerve,	1063
Torticollis or Wry-Neck,	1064
The Twelfth or Hypoglossal Nerve,	1066

Section 9.

DISEASES OF THE MUSCLES.

Myositis,	1068
Myositis Ossificans Progressiva,	1068
Myotonia Congenita—Thomsen's Disease,	1069

INDEX,	1071
---------------	------

HUSBAND'S PRACTICE OF MEDICINE.

CLASSIFICATION.

SECTION 1.—*Morbid States and Processes as such, irrespective of the part or parts affected.*

SECTION 2.—*Diseases of the whole body, and Diseases which may be distributed in several parts of the body, sub-divided as follows :—*

I. Diseases dependent on Specific Poisons — The Infectious Diseases—

1. Acute Infective Diseases of known Bacteriology.
2. Chronic Infective Diseases of known Bacteriology.
3. Septic Diseases of known Bacteriology.
4. Infective Diseases of uncertain Bacteriology.
5. Infective Diseases communicable from animals to man.
6. Diseases due to Protozoa.

II. Diseases due to Worms.

III. Diseases due to Arthropods.

IV. The Intoxications.

V. General Diseases of obscure origin.

SECTION 3.—*Diseases of the Blood, Blood-Forming and Ductless Glands.*

SECTION 4.—*Diseases of the Heart and Blood-Vessels.*

SECTION 5.—*Diseases of the Respiratory System.*

SECTION 6.—*Diseases of the Digestive System.*

SECTION 7.—*Diseases of the Kidneys.*

SECTION 8.—*Diseases of the Nervous System.*

SECTION 9.—*Diseases of the Muscles.*

Section 1.

GENERAL PATHOLOGY.

MORBID STATES OR PROCESSES AS SUCH,
IRRESPECTIVE OF THE PART OR PARTS AFFECTED.

I.—CIRCULATORY DISTURBANCES.

HYPERÆMIA.

Lat. *Hyperæmia*. Fr. *Hyperémie*. Ger. *Hyperæmie*.

An excess of blood in an organ or part is called hyperæmia or congestion. Its blood-vessels are dilated. It may be either *active* or arterial, *passive* or venous, the latter being chiefly due to mechanical obstruction to the outflow of the blood through the veins from disease of the heart or lungs. Active hyperæmia is the result of vaso-motor paralysis, which may be either directly or reflexly produced. As results of active hyperæmia we have increase of temperature, a heightened colour, and some swelling in the part. The dilatation of the arteries does not usually last long enough to be followed by any thickening of their walls. Great cerebral excitability may be noticed. There is also increase in the function of hyperæmic glands, except in those whose functions are only increased by nervous stimulation, the submaxillary glands being taken as a case in point. In the case of venous congestion the tissue elements may atrophy where the increased pressure in the

capillaries takes effect on the surrounding cells. This is well seen in venous congestion of the liver, otherwise called cyanotic atrophy or *nutmeg liver*. The walls of the capillaries are thickened, and the liver cells correspondingly compressed and atrophied. Pigmentation may also occur when blood has escaped from the vessels, as seen especially in the central zone of the liver in the above-mentioned form of cyanotic atrophy of the liver, and in long-standing venous congestion of the lung, where there is also some thickening of the vascular walls, hence the name often given to it of "brown induration." There is also dilatation of the veins with decreased velocity of the blood-current, as in mitral stenosis. Venous congestion may also be followed by hæmorrhage, though this is rare, except in the brain and lungs where the support to the vessel walls is comparatively slight. Severe hæmorrhage may occur in ulceration into varicose veins.

The term "cyanosis" has been applied to all cases where the hyperæmia is due to venous congestion, and in reality means "blueness" of the part, as in cyanotic atrophy of the liver, &c. A cyanosed appearance of the surface of the body is also seen in all forms of suffocation. Cyanosis due to malformation of the heart is treated elsewhere.

Venous hyperæmia may be followed by dropsy.

DROPSY.

Greek ὕδρωψ. Lat. *Hydrops*. Fr. *Hydropisie*. Ger. *Wassersucht*.

DEFINITION—A collection of serous watery fluid in serous cavities of the body, or in the subcutaneous cellular tissue, due to excessive transudation and various obstructive conditions.

Various names have been applied to denote the position of the effusion in dropsy: thus, when in the peritoneal cavity, it is known as *ascites*; in the pericardium, as *hydro-pericardium*; in the pleura, *hydrothorax*; in the ventricles

of the brain, *hydrocephalus*; in the tunica vaginalis of the testicle, *hydrocele*; and when the subcutaneous tissue becomes infiltrated it is called *œdema* or *anasarca*. *œdema* is also used to indicate dropsy of a solid organ, *e.g.*, brain.

Dropsy, as usually met with, is not a disease in itself, but rather a symptom of disease in one or other of the internal organs—*e.g.*, heart, kidney. The composition of dropsical fluid is that of a diluted blood plasma. They both contain water, proteids, and salts. The water in the former averages 96 per cent. or over, and in the latter 91 per cent., while the salts are practically equal in both. The amount of the salts is fairly uniform in all dropsical fluids, which vary chiefly in the amount of proteid which they contain. The position of the dropsical fluid is the chief cause of this variation, though the disease which determines the dropsy has something to do with it. Thus, pleural effusions are generally the most highly albuminous, and peritoneal next, but sometimes it is the opposite. Again, when due to heart disease, dropsical fluid contains more albumin than when due to kidney disease. The question of the causes of dropsy is a complicated one. If theoretical considerations be included it is obvious that dropsy may arise from (1) increased transudation beyond the power of the absorbents to cope with; (2) diminished absorption. Transudation takes place through the capillaries, and it may be increased by (1) dilatation of the arteries and capillaries (hyperæmia); (2) alteration of their walls; (3) alteration of the blood; (4) outside changes in the tissues and nerves of the part. Probably all act by bringing about an alteration in the condition of the walls of the capillaries by which they are rendered more permeable. For an examination of these different factors the reader must refer to a text-book of General Pathology, but we may refer here more particularly to two of the changes in the blood which are regarded as important causal factors, because of their clinical bearing, *viz.*, (1) the hydræmic theory. and (2) the toxic theory. In

anæmic conditions of the blood, dropsies are common, and they have been ascribed to its more watery condition. The result of experiments, however, does not fully bear this out since a simple hydræmia fails to produce dropsy though a hydræmic plethora succeeds. Again, dropsy is a common concomitant of kidney disease, both acute and chronic, although differing considerably in these two conditions. In the former it shows itself first in the loose tissues about the eyes and face, and is usually abundant and quickly produced, pits easily and markedly on pressure, and is often called soft œdema. In the latter it is more obedient to gravity, is slowly produced, and often slight for a long time, and pits less easily, being called hard œdema. The cause differs in the two cases. In the acute form it is doubtless due to the circulation of some toxic substance in the blood acting on the walls of the capillaries. In the chronic forms the toxic substance must be different, as it seems to have very little power in producing the dropsy, cardiac failure and gravity being the chief causal factors. When we come to the second great factor in producing dropsy, viz., diminished absorption, we have much less difficulty. There are only the two absorbent systems in the body, the venous and lymphatic, the former being by far the more important. Complete obstruction of the lymphatics, even of the thoracic duct, is not followed by dropsy, as the veins by increasing their absorptive action are able to compensate for the inactivity of the lymphatics. On the other hand, dropsy may follow upon venous obstruction. In the human subject, œdema of the legs generally follows upon a sudden blocking of the inferior vena cava, although it may not be of long duration; whereas if the femoral vein be experimentally blocked in animals, œdema of the leg does not follow unless arterial hyperæmia be also produced in the limb, *e.g.*, by section of the sciatic nerve. Again, obstruction to the portal vein is commonly accompanied by ascites. It seems hardly necessary to say

that obstruction to both veins and lymphatics will naturally be followed by dropsy. Dropsy will be general when a central lesion involving the whole body is at work, such as heart disease, kidney disease, and anæmia. Many cases of anæmia are secondary to organic disease, but others are primary, being due to the feebleness of the circulation and thinness of the blood; the latter, according to Ringer, is due to a want of albumen in the blood, which reduces the affinity of the blood for the fluids in the parenchyma, and hence these accumulate and produce dropsy. This condition occurs most frequently in chlorotic girls and elderly females. The dropsical effusion is generally in the form of an anasarca. The treatment of this form of dropsy will consist in the administration of one or other of the preparations of iron and stimulating diuretics. The general treatment will be considered when the diseases which give rise to dropsy come under notice in their order.

THROMBOSIS.

Thrombosis is the coagulation of the blood within the vessels during life, especially in the veins where the blood pressure is lowest, and the coagulum is called a thrombus. Embolism is the obstruction of a vessel by a plug transported by the blood-stream from elsewhere, the plug itself being called an embolus. Various forms of thrombi are met with. Red thrombi, consisting mainly of red blood corpuscles with a few fibrin threads and leucocytes, are found when the blood is stagnant; white thrombi, consisting variably of one or other, or combinations of the following constituents, viz., blood-plates, leucocytes and fibrin, are found when the blood is moving; and mixed thrombi are found when white thrombi become fissured, thus allowing

of red blood-cells penetrating into the fissures and coagulating there. The causes of thrombosis are not yet fully determined. Stagnation of the blood plays a most important part, as we find that thrombosis is apt to occur wherever the free movement of the blood is interfered with, as in the eddies which occur in aneurisms, pouchings of the veins, a weak heart, &c. It probably never of itself determines coagulation, as shown by the fact that the blood in a double-ligatured healthy vessel coagulates very slowly, and may remain fluid for days or even weeks. Alteration of the vessel wall is a far more potent factor, and the influence of stagnation of the blood may be in this way explained. This is abundantly exemplified by wounds and inflammations of the vessel walls. When a vessel is ligatured, the internal and middle coats are ruptured and thrombosis ensues. The effect of inflammation in inducing thrombosis is well seen in phlebitis and endocarditis. The integrity of the endothelial lining of the vessels would seem to be essential to the prevention of thrombosis; thus the internal coat of a vessel may be greatly altered, as in atheroma, and yet no thrombosis occur so long as the endothelial layer remains intact. When it becomes impaired coagulation will ensue, and the processes by which it is established are not always the same in every instance. In experimentally-produced thrombosis, for instance, the blood-plates are first seen to collect together and then to become transformed into a homogeneous or granular, firmly-cohering mass. Fibrin and leucocytes may then become superadded, but the proportion in which these three constituents are seen varies in different cases within wide limits. In other instances there is no evidence that blood-plates are necessary, as fibrin threads are formed at once and continue to form the main constituent of the thrombus. We have lastly to consider the influence of the condition of the blood as a factor in causing thrombosis. There would appear to be little room for doubt that

thrombosis may occur in this way, as it has been shown to follow upon apparently unimportant changes in the vessel walls. It has also rapidly occurred in some cases of transfusion where blood from one of the lower animals has been used. Various chemical changes have been suggested in explanation, but definite results have hardly yet been obtained.

The characters of thrombi formed *during life* are important. They are mostly of a dirty white or grey colour, firm in consistence, and distinctly laminated or stratified, and more or less adherent. When formed *after death*, the clot is loose, soft, and easily broken down, not laminated, uniformly dark red if quickly formed, but if more slowly produced, generally shows two layers, an upper pale yellow or buffy and a lower dark red mass. These characteristics will help to diagnose *ante-mortem thrombi* from *post-mortem clots*, and should, therefore, be borne in mind during all inspections for medico-legal purposes. The results of thrombosis as regards the vessel itself and the clot must now be briefly considered.

In the *vessel* the chief changes take place in the inner coat. The middle coat suffers from fatty degeneration and becomes absorbed, and the outer becomes thickened from the increase of the connective tissue. As a result of these changes the elasticity of the vessel is impaired, and there is a tendency to rupture and the formation of aneurism. The walls of the vessel are thickened when the clot becomes "organised"; but should the clot be septic, ulceration may ensue. With regard to the *clot*, it may remain unchanged at the point of formation, or become calcified, or undergo fatty degeneration, either centrally or as a whole. If septic, it breaks down into a pulpy straw-coloured mass, portions of which may be carried into different parts of the body, forming infective emboli. Softening independent of septic organisms, and hence termed simple, is also possible. As a rule, however, the clot becomes "organised," the chief

changes beginning in the walls of the vessel, where they are for the most part found. The clot first becomes adherent to the wall of the vessel, and then the process of organisation begins, and in six or seven days vessels pass from the vasa vasorum into the clot and there form a network. The liquid portions of the clot are absorbed, and the solid undergo fatty degeneration; and lastly, the fibrous tissue contracts on the newly-formed blood-vessels, and the portion of the artery in which the clot was formed is reduced to a fibrous cord. The newly-formed vessels of the thrombus sometimes become very large and sinus-like, largely restoring the obliterated lumen. A softened thrombus frequently breaks down and its fragments are swept away by the blood-stream, forming emboli.

EMBOLISM.

Embolism is due to part of a thrombus or foreign body carried from one part of the circulatory system to another, which it obstructs, and, as a rule, occurs in the smaller arteries and the capillaries. and sometimes in the portal vein. Emboli may be formed by fragments of thrombi, cardiac vegetations, fatty tissue, liver cells, and tumours, or pigment, air, bacteria, or any foreign body. The changes in the embolus and vessel are similar to those in thrombosis. The results are, putting them generally, either increased collateral circulation where such is possible, or, where not possible, degeneration and death of the part normally supplied by the plugged artery. We must carefully distinguish between simple and infective emboli, and though the same circulatory and nutritive disturbances follow in each case, yet in the latter there will be superadded the specific effect of the particular micro-organism present; thus suppuration will follow when any of the pyogenic

organisms are present. It will be necessary, therefore, to confine our attention to the simple emboli. When an artery is plugged by a simple embolus, the changes observed in its area of supply will depend upon the already existing collateral circulation, and the extent and rapidity with which it can be increased. If the radial artery be blocked, the pulse below does not usually disappear, since the ulnar and radial arteries have very free anastomoses in the palm of the hand. If the superficial femoral be blocked, the pulse below the knee disappears, but usually reappears again in the course of a short time. The anastomotic channels between the deep femoral and the articular arteries of the knee are small, and could not of themselves explain this result. Other anastomoses open up freely between the capillaries of the leg and thigh, and thus account for the re-establishment of the circulation and the reappearance of the pulse within a comparatively short time. This capillary anastomosis is of great importance in all cases where the arterial anastomosis is absent or slight, as in the splenic, renal, cardiac, pulmonary, superior mesenteric, and certain of the cerebral arteries, the so-called end arteries of Cohnheim. It is so perfect in the lung that a single simple embolus, plugging a branch of the pulmonary artery, is regularly followed by the development of a collateral circulation, so that no part of the lung tissue suffers. When the emboli are multiple, and plug numerous branches of the pulmonary artery, or when the return of the blood to the heart is impeded, the development of a collateral circulation is, under certain circumstances, prevented and hæmorrhagic infarcts are formed. Similarly in all cases in which local conditions prevent the development of a collateral circulation, the walls of the vessels of the embolised area become more permeable. The blood plasma and some red and white corpuscles escape from them, and more blood flows into them from the neighbouring capillaries or veins. This is followed by stasis in the blood-vessels

of the embolised area, and a free escape of red blood-corpuscles into the tissue of this area. This constitutes the hæmorrhagic infarct. Its nutrition is impaired, and it becomes necrosed. It becomes gradually decolorised, and in course of time may be partially or entirely absorbed, leaving a cicatrix. This condition is typically seen in the spleen. It occurs also in the pyramids of the kidney, and to a certain extent in the heart. On the other hand, if the tissue of the embolised area dies rapidly, and swells up so as to compress the capillaries and prevent not only a collateral circulation but even stasis and diapedesis, it dies rapidly, becoming permanently pale, and forms a white infarct or white softening. The former, the white infarct, is regularly produced in the kidney cortex, after embolism of a branch of the renal artery, as the pale area becomes inspissated and paler; whereas the latter, the white softening, is typically seen in the basal ganglia of the brain when one of its so-called nutrient arteries is occluded by an embolus. These pale infarcts or softenings may also become absorbed.

HÆMORRHAGE.

Hæmorrhage may occur from various situations. Thus we may have hæmorrhage from the nose, *epistaxis* (ἐπι upon, and σταζω I flow, drop by drop); *hæmaturia*, blood from the urinary passages (ἅμα blood, and ουρεω make urine); *hæmoptysis*, spitting of blood (ἅμα blood, and πτυω I spit); *hæmatemesis*, vomiting of blood (ἅμα and εμεω I vomit); *melæna*, blood from the bowels (μελας black, and ἅμα); *metrorrhagia*, blood from the uterus (μητρα the womb, and ρηγνυμι I break forth); *menorrhagia*, synonymous with the last (μην a month, and ρηγνυμι I flow fiercely). The latter is often applied to an excess of blood

at the menstrual periods, the former to losses of blood at other times. Hæmorrhage results from either the direct rupture of a vessel, per *rhæxin*, or the gradual oozing out of the solid and liquid portions of the blood through the walls of the vessels, per *diapedesin* (διαπηδᾶω I leap through).

The two forms of hæmorrhage, however, which more particularly belong to the province of the physician are hæmoptysis and hæmatemesis; and as it is of importance to clearly distinguish the one from the other, we shall give their main points of difference in a tabular form.

HÆMOPTYSIS.	HÆMATEMESIS.
<p>The blood, which is of a bright scarlet colour, comes from the lungs. It is brought up by coughing, is fluid and frothy, from admixture with air. It has an alkaline reaction. Hæmoptysis frequently occurs during the first and last stages of phthisis, and is not of infrequent occurrence in valvular disease of the left side of the heart, and sometimes when there is congestion of the liver with an absence of lung disease. This should be borne in mind.</p>	<p>In this case the blood comes from the stomach, and is expelled by vomiting. It is of a dark purple or black colour (unless a large artery is eroded), more or less coagulated and mixed with the food. It has usually an acid reaction. It may be due to congestion of the liver, cirrhosis, and consequent impediment to the portal circulation.</p>
<p>It accompanies <i>pulmonary apoplexy</i> or infarct, to which reference has just been made under embolism; in it there is extravasation of blood by diapedesis into the air-cells and tissue of the lung, which may produce crepitation in the part till the blood is absorbed.</p>	<p>It may also occur as a vicarious form of menstruation, or from an ulcer or cancer of the stomach, in which latter case it may be followed by <i>melæna</i>, that is, the passage of a portion of the blood with the stools. The blood becomes changed, is very black, and so altered as to look like tar, or even soot.</p>

HÆMOPTYSIS—*continued*

The treatment of hæmoptysis will consist in impressing on the patient the necessity for perfect rest in a semi-recumbent posture, and he must be forbidden to speak. The room should be kept cool. Ice may be kept in the mouth, and a bladder containing ice applied to the chest. If the hæmorrhage be severe, morphine, $\frac{1}{2}$ gr., in the form of *hypodermic injection* should be administered. Cold drinks should alone be allowed, and medicines, as, for instance, the acetate of lead, ergot, gallic or tannic acids, given at frequent intervals.

HÆMATEMESIS—*continued*

In the treatment, ice should also be allowed. If there be much congestion of the liver, a good saline purge, to relieve the portal circulation, will be necessary. Styptic medicines—alum, iron, turpentine, and others—may be given, if the hæmorrhage still continues severe.

INFLAMMATION.

Lat. *Inflammatio*. Fr. *Inflammation*. Ger. *Entzündung*.

DEFINITION—A satisfactory definition cannot be given.

The term inflammation has been so much extended by various authorities since its introduction by Celsus to signify the redness (rubor), swelling (tumor), heat (calor), and pain (dolor) characteristic of superficial inflammations visible to the naked eye, that it is impossible to give a definition which would be acceptable to everyone, and, moreover, the different views are so much at variance that it is unlikely that a unity of opinion will ever be obtained. Some think it ought to be restricted to local disturbances of nutrition accompanied by exudation from the blood vessels, while others make it practically synonymous with local disease. The difference of opinion is not after all of much importance to the practitioner since he may accept

the first and more restricted meaning as holding good for all the naturally occurring inflammations he will be called upon to treat in practice. It is true that there are in man certain tissues, *e.g.*, the cornea, which are anatomically non-vascular, but it is very doubtful if inflammations can occur therein without implicating the blood vessels in their adjacent tissues, and hence they are pathologically really vascular tissues.

The Causes of Inflammation.—These may be summed up as mechanical, chemical, thermic, electrical and infective. The first four act at their site of impact and are often called external irritants. The last may also act in the same way, or it may, without causing change at the point of access, set up inflammation in other parts of the body. Such inflammations are called metastatic and the *noxæ* internal. The infective agents include bacteria and their products. The latter are chemical substances, called, generally, toxins. They are the means by which bacteria produce their effects. They may be formed by bacteria within the body or may be preformed and introduced as such into the body, *e.g.*, in poisonous tinned meats. The name “*ptomaines*” is given to this group of poisons. Injurious chemical substances may be produced within the body independently of bacteria, as in some functional disturbances of internal organs such as the liver and kidney.

The Phenomena of Inflammation.—Disturbance or loss of function first came to be added to the phenomena of Celsus, and later, when the inflammatory processes came to be carefully examined under the microscope, it was seen that other important phenomena were present, which, including as they did the obvious microscopic changes, rendered necessary a re-statement of the changes seen in inflammation. The series of vital processes involved therein may be thus arranged.

1. ACTIVE HYPERÆMIA AND ACCELERATION. — The arteries of the inflamed part dilate, then the veins and capillaries, and the rate of blood flow is increased. The cause is largely a nervous one through a reflex paresis of the vaso-constrictor, or a stimulation of the vaso-dilator fibres, or both. It is not an indispensable process as it may pass off without being followed by other inflammatory phenomena, and these may appear without its preceding them.

2. HYPERÆMIA AND RETARDATION. — The dilatation of veins and capillaries increases. The blood flow becomes slower until it is slower than normal. The distinction between axial stream of corpuscles and peripheral stream of plasma becomes lost, and the lumen becomes filled with a mass of red and white corpuscles which move slowly onwards for a time and ultimately come to a complete standstill—a condition of stasis. The corpuscles, especially the white, stick to the vessel walls. The cause is the alteration of the vessel walls and surrounding tissues produced by the action of the irritant.

3. ESCAPE OF FLUID. — There is a liquid transudation from the blood vessels, particularly the capillaries and veins, which differs from the normal lymph in amount and composition. It is more abundant and contains a greater amount of solids, particularly albumen. A deposit of fibrin sometimes occurs. It is not a mere filtration but a true secretion, the vascular walls retaining and exercising their secreting powers. The chief cause is alteration in the vessel walls, though the increased blood pressure may assist it. The exudate always contains cells, and if it be very rich in leucocytes it is called pus, if in red corpuscles it is hæmorrhagic. The greater the quantity of the exudate, the greater will be the severity and duration of the inflammation. It may act a beneficial part when serous or watery, by flushing out the injured area and diluting the irritant, and when fibrinous, by circumscribing the inflammation,

as in many cases of appendicitis. It may also help in the destruction of bacteria and in assisting the nutrition of the tissues; but it plays a distinctly hurtful part when it collects in the serous cavities of the body, the ventricles of the brain, the alveoli of the lung or the air passages, or when it forms a favourable nidus for bacterial growth and development.

4. THE ESCAPE OF BLOOD CORPUSCLES.—(1) *The Leucocytes*.—The first step is their margination along the wall of the altered vessels, especially the small veins. Then they become adherent and push a portion of their protoplasm through the vessel wall, gradually following it by the rest of their bodies. It is chiefly the polymorpho-nuclear leucocytes, comprising roughly 70 per cent. of the total number of the leucocytes present in the blood, which emigrate. They pass out by means of their amœboid movement, which is largely dependent upon the nature and intensity of the irritant. It has been suggested that substances having an attraction for the leucocytes are formed in the tissues in inflammation. This is referred to as positive chemotaxis. The products of bacteria and of the tissue degenerations may be the sources of such substances. Positive chemotaxis is an important stimulant of amœboid movement but it is not its cause, as leucocyte emigration may occur when it is absent. Leucocytes emigrate most abundantly in suppurations, and collect chiefly around the cocci, many of which they engulf. It is supposed that the cocci are destroyed in this way. The leucocytes are consequently called phagocytes, and the process phagocytosis. It is the polymorpho-nuclear and the large hyaline uninuclear leucocytes which act as phagocytes. It has been asserted that phagocytosis is always a beneficial act, and certainly it is so when it causes the destruction of active and vigorous germs, but it is not so when it encourages and assists, as it sometimes does, the

development of the bacteria and their transference to healthy situations where they may set up new foci of disease. Leucocytes will ingest solid particles of all kinds, *e.g.*, fat, pigment, red cells, &c., as readily as germs, and phagocytosis may be fundamentally of a nutritive nature. Other leucocytes break down and may be the source of fibrin ferment, of the bactericidal and many other constituents of the fluids of the body. Pus corpuscles are formed entirely by leucocytes. Metschnikoff and others hold that leucocytes may also be transformed into fixed tissue cells, but, while the possibility cannot be denied, it has never been proved to occur in the tissues of the higher animals at any rate. (2) *Escape of Red Cells*.—Unlike the emigration of leucocytes, this is a purely passive process. It is called diapedesis and is due to the alteration of the vessel walls, aided by the increased density of the blood and the blood pressure. (3) *Escape of the Blood Plates*.—They escape like the red cells, but few observations have so far been made upon the process.

5. THE ROLE OF THE FIXED TISSUE CELLS.—They show two stages:—(1) Degenerative and (2) Regenerative. The degenerative changes follow more or less quickly upon the action of the irritant. Cloudy swelling, fatty and other degenerations are observed in the endothelial cells and other tissues. They may also become detached and later may proliferate. It is difficult to say exactly where the degenerative changes end and the regenerative begin, but proliferation probably always belongs to the latter stage. The proliferated cells may afterwards produce formed tissues which replace those destroyed by the irritant. The newly formed tissues are generally like those which they replace, but are liable to be of an inferior character in the more highly endowed structures. Thus connective tissues in the main replace lost structures in the central nervous system, liver and kidney. The blood vessels enter

into the process of regeneration wherever there has been loss of tissue. In an ulcer, which may be taken as the type, the floor becomes covered with little red points, known as granulations. Each consists of a vascular loop surrounded by leucocytes and connective tissue cells. The latter are often called epithelioid. They give rise to new fibres and gradually the gap becomes filled up with a fine vascular fibrous tissue. The surface becomes at the same time covered by epithelium formed by the proliferation of the pre-existing surface epithelium. The new blood vessels arise as buds from the pre-existing capillaries. These conditions hold good for deep (muscles, bones, brains, &c.) as well as for superficial inflammations. Considerable transformations may afterwards occur in newly formed tissues. The capillaries may largely disappear and the fibrous tissues increase greatly. The ultimate cicatricial tissue is often denser, less elastic, and more vulnerable than the original tissue. The fixed tissue cells show still other changes. They may act as phagocytes, being known as macrophages to distinguish them from the leucocytes which are called microphages.

NOMENCLATURE USED IN INFLAMMATION.

1. *Terms signifying Inflammation.*—The affix *itis* added to the Greek name of an organ indicates an inflammation thereof—*e.g.*, peritonitis, meningitis, &c. The prefixes *peri* and *para* placed before these terms indicate that the serous covering of the organ and the surrounding connective tissue respectively is inflamed—*e.g.*, perihepatitis, parametritis, &c.

2. *The Duration of Inflammation.*—It is acute, sub-acute, or chronic, according to the length of its course.

3. *The Kinds of Inflammation.*—A satisfactory classification cannot be given, but various groupings are in use. Thus they are spoken of as superficial and deep, according to their site; as parenchymatous and interstitial, according

as they attack the active cells of an organ or its supporting connective tissues; as serous, purulent, fibrinous, or hæmorrhagic, according to the nature of the exudate.

II.—NUTRITIONAL DISTURBANCES.

NECROSIS.

DEFINITION—Local death of tissues within the living body.

The causes of Necrosis are—(1) Mechanical; (2) Extremes of temperature; (3) Chemical; (4) Arrest of nutrition; (5) Toxic and infective; (6) Nervous. That charring, crushing, or corrosive fluids may cause necrosis is obvious. Arrest of nutrition may be brought about by many circulatory disturbances, *e.g.*, ligature, embolism, thrombosis. It need not be permanent. A very short arrest will suffice to produce necrosis in very sensitive and highly specialised tissues, *e.g.*, the brain. Lowered vitality of the tissues, such as occur in old age, &c., and in certain troubles of innervation, are very important pre-disposing agents, so that a comparatively trivial arrest may prove effectual when in a healthy individual it would be harmless. Toxic causes are illustrated by such drugs as ergot, and the infective by micro-organisms in general, many of which, by means of the poisons which they excrete, have a great power of causing necrosis. The nervous causes are illustrated by bedsores and perforating ulcer of the foot, though there is probably a more complex mechanism at work than the nerve influence alone.

The forms of Necrosis are—

1. Coagulation necrosis.—This occurs in cellular tissues and those rich in protoplasm when rapidly deprived of nutrition, &c. The cells die and produce a nucleo-albuminous substance which reacts upon the coagulable lymph

coming from without, and produces a coagulation within the cell. The affected part comes to resemble coagulated fibrin and stains badly. Examples are seen in white infarcts, atheroma, &c. The vitreous degeneration of muscle described by Zenker, in which certain muscular fibres become hyaline, is merely a variety of this form of necrosis.

2. Colliquative Necrosis.—This occurs when the dead tissues become infiltrated with a serous fluid. It is seen characteristically in the brain (embolism), the cerebro-spinal fluid (the lymph of the central nervous system) being non-coagulable.

3. Fat Necrosis.—It occurs in the pancreas, associated with acute pancreatitis, and is probably caused by bacteria.

4. Caseation.—This is merely a mode of termination of necrosis. The dead tissues lose fluid and become dried up into a cheesy-like mass.

5. Gangrene.—This is necrosis, plus putrefaction, and it only occurs when the putrefactive organisms can gain free entrance into a dead tissue, and therefore is seen in parts exposed to the air. Two forms are recognised—moist, in which the conditions are favourable to the growth of the putrefactive germs; and dry, in which they are not. In the latter the part becomes dense and hard, shrivelled and brown or brownish-black (mummification). It is apt to occur in old people with atheromatous arteries and weak hearts. A thrombosis generally occurs in the arteries of the leg, and gangrene of the foot follows. It is a chronic process and usually of limited extent; a zone of inflammation (the line of demarcation) forming between the living and dead parts. In moist gangrene similar changes in colour appear; the epidermis is raised into bullæ and blebs and the odour is very foul. The process is generally more rapid and tends to spread, hence early amputation is usually wise.

ATROPHY.

Lat. *Atrophia.* Fr. *Atrophie.* Ger. *Atrophie.*

The term atrophy is applied to any diminution in size of an organ or the body as a whole. It is called simple if the diminution in size is not due to degenerative processes, and degenerative when so due. Simple atrophy is caused either by a diminution in the size or number, or both, of the elements of the tissue, the essential microscopic structure being unchanged. Sometimes the protoplasm of the atrophied cells shows much pigmentation of new formation. This is known as pigmentary atrophy. Simple atrophy may be either local or general; in the former the subcutaneous adipose tissue first disappears, then that in the deeper parts. Defective nutrition, both in quantity and quality, deficient vascular supply, pressure and disuse or cessation of the normal functions of a part are the causes at work in the production of atrophy, either general or local. Defective nutrition, as a result of defect in the quantity of nutriment taken, is seen in cases of starvation, the result of disease or accident; in children, from the administration of improper food. Atrophy from deficient vascular supply may be seen in the testis, when from any cause its blood-supply is diminished. Atrophy from pressure may be seen in bones as the result of the pressure exerted by aneurisms or other tumours, and even in some organs, as in the kidney, for instance, by pressure exerted by some abnormal growth of one or other of its normal constituents. The effect of disuse is seen in the stiffened outstretched arm of certain Indian devotees or in ordinary paralytic affections of the limbs. Hence we speak of atrophy from disuse, pressure atrophy and neurotic atrophy. The condition of the central nervous system is of great importance in relation

to the nutrition of the body; thus destruction of the ganglion cells in the anterior cornu of the spinal cord is followed by atrophy in the muscles, bones, ligaments, tendons, and skin of the extremities, as in infantile paralysis, &c.

DEGENERATION (CLOUDY SWELLING).

Lat. *Degeneratio*. Fr. *Dégénérescence*. Ger. *Degeneration*.

DEFINITION—Alteration in the *quality* of a tissue either due to chemical or molecular changes of its protoplasm, so that by such alteration there is a tendency to ultimate destruction or death of the tissue, preceding impairment of function.

Degeneration differs from atrophy in this, that nutrition in the former is altered in *quality*; in the latter, in *quantity*. As a result we have in atrophy waste of tissue in excess of the assimilation of new material; in degeneration a metamorphosis of the tissue and an abnormal substance exists which is not consumed. Thus the vitality and functions of the tissue are impaired. The functions may be fairly well carried out at first, but even then they are always impaired. The terms "parenchymatous" or "granular dégénération," "albuminous infiltration," "molecular degeneration," or, lastly, "cloudy swelling" (by Virchow), are applied to the first changes in commencing degeneration. It specially affects cells whose specific function is chemical, notably the cells of the glandular organs, the liver and kidney, also cells lining mucous membranes and muscle cells, particularly the muscle fibres of the heart, but also other muscular fibres. The causes at work in the production of "cloudy swelling" are (1) high temperature, (2) many poisons, *e.g.*, arsenic, phosphorus, carbolic acid, &c., (3) interference with nutrition, and (4) most strikingly by the action of certain organisms, particularly the pyogenic organisms. Hence it occurs in many infectious diseases,

e.g., pyæmia, septicæmia, typhoid. To the naked eye, as in the liver, where the change is generally most marked, the organ appears somewhat enlarged and anæmic, and of a dull opaque greyish-white colour. Examined under the microscope the cells are swollen and granular, and in some cases contain clear albuminous globules and fatty particles. The granules are very numerous and small, obscuring the nucleus. They are probably albuminous in nature. The swelling of the cell is due to the absorption of water. In some cases the organs attacked recover their normal appearance; in others, fatty degeneration or necrosis supervenes.

In order to distinguish "cloudy swelling" from other degenerations, the following table may be of use:—

1. The granules are cleared up by acetic acid (1% solution.)	}	These show that the granules are not fatty in nature.
2. Not affected by ether or chloroform.		
3. Not stained black by perosmic acid.		
4. The granules are not stained by carmine or logwood.	}	This shows that the granules are not composed of protoplasm.

FATTY DEGENERATION.

Lat. *Adiposa*. Fr. *Graisseuse*. Ger. *Fettige*.

DEFINITION—A process by which there is a gradual replacement of the protoplasmic elements of a part by molecular fat. It is a retrogressive change, similiar to cloudy swelling.

Due to certain causes, to be presently mentioned, the normal protoplasmic constituent of the tissues is so changed that its molecules are converted into fat. It is, in fact, a transformation of the protoplasm into fat, and thus differs from "fatty infiltration," in which the cells take up

into their substance particles of fat which do not form their normal constituent. In the latter case, the cells may again become healthy by parting with the accumulation of fat in them; in degeneration, if of high degree, this cannot take place, as it is the cells themselves that have undergone change, and it may lead to their complete disintegration into fatty and albuminoid molecules, but a slight or moderate degree may recede and recover.

Fatty degeneration may be either *physiological*, as is the case in the uterus after delivery, the secretion of milk, &c., or *pathological*, as in the heart, liver, kidney, and other organs. The results of fatty degeneration are flabbiness, and brittleness of the part, decrease in its weight and specific gravity, atrophy and a change of its colour to a greyish-yellow or brownish-yellow tint. Under the microscope the cells of the affected part may be seen swollen at first, and in acute cases, but, as a rule, they are shrunken and wasted and full of oily granules, generally numerous and of small size, not displacing the nucleus.

Fatty degeneration occurs most frequently in highly organised tissues, indeed in much the same tissues as cloudy swelling. It therefore occurs in all parenchymatous cells, particularly of liver and kidney; also in muscles, particularly the heart; and in nerve cells, fibres, &c. The presence of fat may be detected by the granules being soluble in ether or chloroform, unaffected by acetic acid and blackened by osmic acid. The causes at work in the production of fatty degeneration in the tissues are such as bring about *local* or *general* metabolic disturbances.

The *local* may be caused by (1) disturbances of the circulation which limit the supply of nourishment, *e.g.*, embolism, thrombosis, ischæmia, &c.; (2) increase of function requiring a greater increase of nourishment than can be supplied by assimilation from the blood; (3) the local action of poisons, *e.g.*, the pyogenic toxins around abscesses.

The *general* disturbances may be classified much in the same way:—(1) Insufficient supply of nourishment. The tissues require a sufficient supply in order to enable them to maintain their normal functions. If it be insufficient, the albumen of the tissues is called upon to make up the deficiency. This is seen in marked degree in starvation. The tissues at first simply lose weight from their albumen being used up, but later fatty degeneration appears, especially in liver, kidney, &c. It is also seen in all anæmias, such as pernicious anæmia, leukæmia, &c., and in such cases it is often exaggerated by the superposition of certain toxic influences which may prevail in these conditions. (2) Increase of function. This is met with in pyrexia. A simple hyperthermia may of itself cause fatty degeneration by increasing function and thus the demand for nutriment. In man, pyrexia is most frequently caused by infection, when the influence of the specific toxins becomes super-added in this direction. (3) Intoxications. Here the poisons act generally through the circulation. Phosphorus, alcohol, arsenic and carbonic oxide may be cited as examples. They probably act through the increased disintegration of albumen accompanied by a diminution of the process of oxidation. The specific poisons generated by bacteria are still more frequent causes. They may act more upon certain organs than others, notably the kidney, on account of their accumulation in these organs after their absorption by the circulation.

MUCOID DEGENERATION.

Lat. *Mucosa*. Fr. *Muqueuse*. Ger. *Schleim*.

DEFINITION—The transformation of the albuminoid *constituents* of the tissues into mucin, a reversal of the foetal process in which the higher is developed from the lower.

As the development of the body proceeds, the primary tissues, except in the case of the *vitreous humor*, which is permanently a mucoid tissue, become developed into higher forms of connective tissue. In mucoid degeneration the degenerative process is closely allied to those processes normally occurring in the foetus, only reversed; the tissues becoming converted into a soft mucilaginous jelly-like substance, giving the reactions of mucin. It differs from albumen in not containing sulphur. It is precipitated from weak alkaline solutions by acetic acid if no neutral salts, particularly sodium chloride, be present. It is also precipitated by alcohol. It is not precipitated by boiling, by corrosive sublimate, or by tannin. As a physiological process it occurs in the secretion of mucus in the mucous membranes of intestine, &c., and many glands. It also occurs in the form of small drops in the cells lining mucous membranes in catarrhal conditions.

Mucoid degeneration may occur in cartilage, especially in cartilaginous tumours, where the matrix first becomes soft. Hyaline drops then appear in the cells, and finally these also break down and a homogeneous mucoid substance is formed. It occurs similarly in cancers, fibromas, lipomas, myxomas, myomas, &c., and in many diseased organs.

COLLOID DEGENERATION.

Lat. *Colloides*.Fr. *Colloïde*.Ger. *Colloid*.

DEFINITION—A form of degeneration in which the albuminoid *contents* of the cells are converted into a peculiar “colloid” material, differing from “mucin” in containing sulphur and not being precipitated by acetic acid or alcohol.

The colloid material is colourless, or yellow translucent, has the consistence of half-set glue, and first makes its appearance as small lumps, chiefly *within* the epithelial cells, which it ultimately fills. It chiefly, and probably only, affects epithelial cells. The cells in time coalesce to form larger masses of jelly-like material, readily visible to the naked eye. The cells are destroyed, the intercellular tissues atrophy, and cavities containing the colloidal substance are formed. Its precise chemical composition is not yet known, but much important work in this direction has been recently done by Dr R. Hutchinson and others. Colloid change is met with in the thyroid gland as a physiological condition, but it may be increased as in colloid goitre, when it probably passes the physiological boundary and becomes pathological. It is also seen in the prostate gland and perhaps also in the tubules of the kidney where a substance closely resembling it is found as casts. It is, however, most characteristically seen in certain new growths, notably in multilocular ovarian cysts, where the cells lining the spaces are often so full of colloid material that the name colloid cystoma is a common synonym for the tumour. It is also present characteristically in a certain form of cancer, the colloid cancer, which is especially common in connection with the gastro-intestinal canal.

HYALINE DEGENERATION. ∴

DEFINITION—The transformation of tissues into a clear, firm, glassy, translucent tissue, differing from waxy degeneration, of which it may even be an earlier stage, by certain staining reactions only.

This is a change which is not yet clearly understood, and it will be necessary only to treat of it very shortly here.

Many examples of its occurrence can be given, and perhaps the most striking is that which occurs in the arterioles and capillaries of the spleen, lymph glands, &c., in certain diseases, *e.g.*, diphtheria. It would appear to begin in the inner coat, and then to extend to the middle or even outer coat. It affects the vessel, often irregularly. The affected part is clear, glassy and translucent. It stains faintly with red and blue stains, but does not give the characteristic rose pink colour of waxy degeneration with methyl-violet. It is also seen as hyaline casts in the kidney, about whose origin various views are held. Other examples of it are hyaline thrombi, and the clear, translucent degeneration which may occur in the stroma of lymphatic glands and of certain tumours. Its occurrence in diphtheria suggests that a ferment is probably instrumental in its production.

LARDACEOUS DEGENERATION.

(ALBUMINOID, AMYLOID, WAXY.)

Lat. *Lardacea*. Fr. *Lardacée*. Ger. *Speckige oder amyloide*.

DEFINITION—A degenerative tissue change, probably the result of their altered nutritive properties, by which altered proteid substances circulating in the blood pass out of the blood into the tissues, where it forms the waxy substance by the action of the cells.

Lardaceous, albuminoid, or amyloid degeneration is one of the most important of the tissue changes. It may be found in every tissue and organ, but the organs most frequently attacked are the liver, spleen, kidneys, intestine,

supra-renal bodies and lymphatic glands. It seldom occurs in the muscles and brain. The *Corpora Amylacea*, found normally in the central nervous system in the neighbourhood of the ependyma of the ventricles and also in the brain and spinal cord within diseased foci, are supposed to be masses of waxy matter, but their exact chemical composition is unknown. They give much the same reactions as the lardaceous material found in other organs. The conditions under which waxy disease most frequently occurs are in cases of prolonged suppuration, chronic phthisis, and constitutional syphilis. The parts affected are first the middle coats of arterioles, then the walls of capillaries external to the endothelium. It also affects the middle coats of larger arteries and connective tissue fibres generally. The altered tissues are swollen and semi-translucent, with a peculiar, homogeneous, glassy, or waxy appearance, and with a dilute solution of iodine give the characteristic mahogany brown colour under reflected light and a golden-yellow with transmitted light. This is a very characteristic and delicate test, but perhaps the best microscopic test is methyl-violet. This stains the waxy parts a brilliant red colour and the healthy tissues a blue-violet. The amyloid material—once regarded as a starchy substance, hence the term “Amyloid Degeneration”—differs from the starches in containing nitrogen, and is not, as suggested by Dickinson, dealkalised fibrin, its chemical constitution being against that theory. It contains the same amount of nitrogen as albuminoid bodies and is perhaps a decomposition product of albumen, to which it stands in close relation. It differs from albumen in its insolubility in water and its resistance to alkalies and acids and also to the gastric juices. Waxy degeneration may end in resolution or fatty degeneration, rarely calcification, and in necrosis resulting in the formation of a mass of granules which do not give the waxy reactions.

During life the presence of this form of degeneration may be suspected in cases of tertiary syphilis, chronic

phthisis and long-continued suppuration connected with bone disease. The organs affected increase slowly in size, become dense and firm with a mottled translucent appearance; and their functions are more or less impaired as the degeneration increases. There is considerable evidence to support the view that waxy degeneration is caused by a ferment formed especially by the pyogenic organisms, but exact proof is still wanting.

FATTY INFILTRATION.

DEFINITION—A condition in which the cells of a part become abnormally loaded with fat, but the tissues themselves remain, for a time at least, normal.

The fat globules are deposited in the otherwise unaltered cells. The protoplasm of the cells shows no degenerative or disintegrative change, whereas in fatty degeneration the protoplasm of the cells in which the fat globules are seen does show these changes. This is a much more reliable test than the size and number of the oil globules present. These, in the liver, for instance, are generally said to be larger and fewer in number in infiltration than in degeneration, but the preservation of the physical and chemical character of the protoplasm of the cells in the former is a much more reliable distinction. Again, in fatty infiltration the fat comes partly from an increased supply of fat to the cells, brought to them by the circulating blood, but it also comes partly from an excess of circulating albumen which is withdrawn from the blood by the cells and formed into fat within their protoplasm. In fatty degeneration, on the other hand, the fat comes from a destruction of the organised albumen of the cell protoplasm itself. Fatty infiltration is, within certain limits, a purely physiological condition. It is only when it reaches a very high degree that it becomes

pathological. The causes of the latter are both predisposing and exciting. The former are very important. There is, for instance, an individual predisposition to the condition which is often hereditary in families. The exciting causes are (1) excess of food. The excess may consist either of fatty or mixed food. The fat is absorbed from the intestine into the blood, in which it is usually kept liquid by the albumen and salts of the plasma, but in marked cases it may be seen therein in the form of minute droplets. The excessive albumen of the mixed diet is also absorbed from the intestine into the blood. The fat is withdrawn from the blood by the cells, and so is the albumen, and formed in situ into fat. It has now been abundantly proved, experimentally and otherwise, that protoplasm has the power of bringing about this transformation. (2) Insufficient bodily exercise. This is of considerable influence in supporting over-feeding, as it favours the accumulation of fat through deficient oxidation, &c. (3) Alcoholic indulgence. This acts much in the same way. These causes explain a general fatty infiltration or lipomatosis universalis, or adiposity, or obesity, as it is often called. There also exists, however, a local lipomatosis. It is especially seen where an organ or portion of an organ has atrophied, as between the atrophied muscular fibres of the heart, around the pelvis of a contracted kidney, and between the atrophied muscular fibres of the skeletal muscles. Such muscles, however, always, with the single exception of pseudo-hypertrophic paralysis, look thinner than normal, notwithstanding this deposition of fat. The deposition of fat in this pseudo-hypertrophic muscular atrophy is more striking than in any other form, and causes the individual muscles, especially of the calf, to appear, for a time at any rate, to be more or less considerably increased.

CALCAREOUS INFILTRATION.

Lat. *Calcareo*. Fr. *Calcaire*. Ger. *Kalkföunige*.

DEFINITION—An infiltration of the tissues with calcareous materials.

Simple calcification must be distinguished from ossification. The latter is an *active* process in which the lime infiltration is an essential part of the new formation of tissue; while the former is essentially a *passive* process, in which no new tissue is formed. The calcareous particles, chiefly lime and magnesia salts, as phosphates and carbonates, occur both in the cells and intercellular substance of the part, and are seen as fine, refractile granules, appearing dark by transmitted light, but white by direct light. When viewed under the microscope the tissues are generally more or less opaque. Mineral acids, such as hydrochloric, dissolve them with evolution of CO_2 if much carbonate present. Calcification occurs as a physiological process in cartilage, *e.g.*, the costal cartilages of old people. It is seen most characteristically as a pathological process in dead tissues, *e.g.*, diseased parts of vessel walls, thrombi (phleboliths), dead embryos retained in the abdomen or uterus (lithopædion), dead parasites, &c. These probably occur only when large quantities of lime salts are taken up by the blood.

 PIGMENTATION.

Lat. *Pigmentaria*. Fr. *Pigmentense*. Ger. *Pigment*.

DEFINITION—An infiltration of pigment in the tissues.

There are three sources of pigmentation in the human body:—(1) The blood pigment, hæmoglobin. (2) The non-pigmented nutrient fluids. This is autochthonous pigmentation. (3) Extraneous sources, pigmentation by importation.

I. THE BLOOD PIGMENT.—The principal derivative of the blood pigment is bile pigment or bilirubin. (1) The bile pigment may accumulate in the blood, either from increased absorption by the bile ducts and intestines or from increased formation of bile pigment and its subsequent passage into the blood. This causes general icterus or jaundice. The bile pigment is present in solution in the tissues. There are four chief forms of jaundice—(a) obstructive, which results from occlusion of the bile ducts, especially the common bile duct, as is seen so frequently in man in catarrhs of the duodenum; (b and c) toxic and infectious, in which special poisons (introduced from without in the toxic and formed by bacteria in the body in the infectious) cause solution of the red blood-corpuscles and thus an increased formation of the bile pigments within the liver. Thence they are absorbed in large quantities by the walls of the bile ducts and intestines and thus pass over into the blood; (d) jaundice in the newly born, icterus neonatorum. In many cases there is occlusion of the bile ducts, probably the result of hereditary syphilis. In some cases it may be infective through septic infection from the umbilical cord. (2) Bile pigment is also formed in connection with local hæmorrhages, *e.g.*, after a black eye the phases of colour are partly due to the formation of hæmatoidin, now proved to be identical with bilirubin, which is gradually absorbed. In blood extravasations, generally within the tissues, two pigments are derived from the hæmoglobin of the blood, *viz.*, hæmatoidin and hæmosiderin. The former generally forms dark brown rhombic crystals, but sometimes granules; it contains no iron; is insoluble in water, alcohol and ether; soluble in chloroform, bisulphide of carbon and alkaline fluids. The conditions which determine its formation are not accurately known. It would seem to be formed when there is a deficient supply of oxygen, and it may be that some ferment is required. The latter occurs as yellowish-brown, brightly-coloured, rounded granules

and clusters of granules, which are insoluble in water and give marked iron reactions. Thus they blacken with ammonium sulphide, and become blue after a mixture of sulphuric or hydrochloric acid and potassium ferrocyanide. Hæmosiderin, therefore, agrees completely in these reactions with hydrated oxide of iron. Its formation would appear to take place only in the presence of a free supply of oxygen. (3) Melanin pigment. This is a dark brown pigment which is formed from the hæmoglobin of the red blood-corpuscles by the action of the plasmodium malariae. It is at first formed within the body of the parasite residing inside the red corpuscles, but is subsequently set free in the blood-plasma, when the parasite sporulates and escapes. It is also found in varying quantity in the internal organs, such as the liver and spleen.

2. AUTOCHTHONOUS PIGMENTATION.—Here the pigment is formed by the tissues from the non-pigmented nutrient fluids. It occurs either as granules or in solution. The former is seen physiologically in the pigmented layer of the retina and skin, and pathologically in the skin and mucous membranes in Addison's disease, as well as in new growths, such as pigmented moles of the skin and melanotic sarcomata. In solution it is seen in the yellow colouring matter in the cells of the corpora lutea (physiological) and in the fat cells of a skin tumour called xanthelasma (pathological).

3. EXTRANEOUS SOURCES.—This is seen in the skin in tattooing, and in the lungs in inhalation of certain dusts, *e.g.*, anthracosis, from soot and coal dust, siderosis, from the hydrated oxide of iron or English-red. It is also seen in general argyria caused by absorption of silver from the intestines, and as a local argyria in those who work among silver.

HYPERTROPHY.

Lat. *Hypertrophia*. Fr. *Hypertrophie*. Ger. *Hypertrophie*.

An increase in size, a uniformity in the character of the normal constituents of an organ or tissue resulting from various causes, but chiefly from excessive nutrition, is known as hypertrophy. The form, structure, and functions of the organ are at the same time preserved. This condition is seen in hypertrophy of the heart subsequent to valvular disease; in the kidney when its fellow has become diseased. When the increase in the size of an organ takes place as a result of an overgrowth of some interstitial tissue, loss of function, as a rule, occurs, and this increase in size is described as *false* or *pseudo*-hypertrophy, and is well seen in the disease known as pseudo-hypertrophic paralysis. The term pseudo-hypertrophy is, however, not often used, and is practically confined to this disease, in which the apparent hypertrophy is due to a local lipomatosis or formation of fatty tissue between the atrophied muscular fibres. Hypertrophy may be the result of an increase in the size of the individual elements, or in the number of the elements of an organ; in the former case it is said to be *simple* or *true*, in the latter *numerical*. To this latter form the term *hyperplasia* has been given. In most cases of hypertrophy the two forms are combined. Mere increase in the size of an organ, it must be borne in mind, does not constitute genuine hypertrophy. A waxy liver, although increased in size, is really the subject of atrophy, for its true hepatic or secreting tissue is decreased. The following are the conditions under which hypertrophy may occur. It may be compensatory or functional, as in the case of an increase in the myocardium from valvular disease, or in the loss of one kidney, either by disease or operation. In the case of the heart the hypertrophy is due to the increase in work done by the heart, hence the term functional;

and, further, since it enables the heart to overcome the valvular resistance and to maintain the circulation in a normal condition, it is called compensatory. The same explanation holds good for the kidney. The loss of renal tissue is quickly made good, but this is best secured when the loss occurs early in life. Thus, in congenital absence of one kidney the other may be double the normal size or even larger. It may be stated as a general rule that young tissue is capable of a much greater hypertrophy than adult tissue. Compensatory hypertrophy occurs in other organs as a result of increased function, as in one lung, one testicle, one ovary, &c., in cirrhosis of the other. Compensatory hypertrophy is not, however, always due to an increase of function. Thus, in newly-born animals, if one testicle or ovary be removed the other will hypertrophy. It may possibly be due to some influence of the central nervous system. The causes of hypertrophy may be thus summed up: excessive nutritive supply, either as regards the quantity or quality of the supply; increase of function or overwork, seen in the voluntary muscles—the overwork must not, however, be excessive; irritation, applied to growing parts, probably by increasing the vascular supply, as seen in the lengthening of a long bone, of which one epiphysis remains ununited. Hypertrophy is also caused by intermittent pressure, seen in the growth of the cuticle in corns on the feet and hands.

NEW GROWTHS.

DEFINITION—So closely are new growths or tumours connected with hypertrophy and various inflammatory new formations that a satisfactory definition cannot be given.

A tumour is usually regarded as a more or less circumscribed growth of a part which shows by pressure or destruction of the normal structures of the part that it is

only partially subject to the laws which govern the part itself. Tumours tend to reproduce the structure of the part in which they arise, although they sometimes differ considerably therefrom.

Classification.—The most satisfactory classification is a histological one, although the clinical division into simple, or benign, and malignant is a most convenient one. The simple tumours consist of tissues which are identical in structure with the normal physiological tissues of the body. They are usually circumscribed and do harm only by their pressure effects and interference with function, but some occasionally take on a rapid growth and other characters which we look upon as malignant. The myxoma, glioma, lipoma, fibroma, myoma, chondroma, osteoma, angioma, papilloma and adenoma are examples of simple tumours. The malignant tumours, on the other hand, grow more rapidly and infiltrate the tissues around them, giving rise to new growths like themselves, either in the neighbourhood of the primary growth or in distant parts of the body. They are divided into Sarcomata, which are mainly cellular tumours with a relatively scanty stroma or matrix mixed up among the cells, and blood-vessels of a rudimentary character lying in contact with the cells and consequently tending to spread by the blood-vessels; and Carcinomata, which are epithelial tumours (epiblastic or hypoblastic), growing as cell clusters or groups surrounded by fibrous tissue, giving an alveolar formation. The fibrous tissue thus surrounds groups of cells and does not lie among the individual cells. The blood-vessels lie in these septal walls and not in contact with the cells. The lymphatics also lie in the septa but communicate freely with the alveoli, and hence these growths tend to spread, not by the blood-vessels, but by the lymphatics.

Etiology of Tumours.—This still awaits a satisfactory explanation. Various theories have been put forward which may be shortly thus arranged:—

1. PREDISPOSING CAUSES.—Many facts, such as the prevalence of cancer in certain families, go to show that the constitutional factor plays an important part, possibly as important a part as it is now universally admitted to do in tuberculosis. It may be a hereditary weakening of resistance of the tissues, or even an actual presence of some infective virus in a latent condition, which makes them an easy prey to the real agent.

2. EXCITING.—Of the various suggested theories it will suffice here to notice two—the embryonic residue theory and the irritative one. The former assumes that more cells may be formed than are required for the formation of a part during its development, and that these cells, although they may remain long dormant, yet may at any time, since they retain their power of multiplication, become active and by their increase give rise to a tumour. The central enchondroma is a good example. In the same way it is assumed that such embryonic residues may in the course of development wander from their own site into a neighbouring organ, and subsequently give rise to a tumour there, as in certain tumours of the kidney which resemble the suprarenal body in structure. The latter, the irritative, theory is illustrated by the frequent occurrence of epithelioma of the lower lip in those who use a short clay pipe, in the occurrence of mammary cancers after irritations of the nipple, in the frequent occurrence of cancer at the various ostia of the gastro-intestinal canal, and in the occurrence of cancer in the scrotum of chimney sweeps, usually attributed to the irritation of the soot, and to an exactly similar form which occurs among workers in paraffin factories. By far the most important element in irritation is the possible entrance of an infective agent at the site of injury. Coccidia-like bodies, mostly circular in shape, with a nucleus, can be easily demonstrated to be present within the cells of a cancer; and two very different

views are at present held as to their nature. Many consider them to be true parasites belonging to the protozoa, and some of those who hold this view go further and look upon them as the true cause or infective agent of cancer. If this should in future be proved, then we may, reasoning from analogy, suggest that some similar cause is at work in the production of all tumours; and then it is obvious how important it will become to guard against all initial injuries and catarrhs, which may afford a means of entrance for these germs. The other view however, supported by a strong weight of evidence, regards these coccidia-like bodies as merely accidental cell inclusions and nuclei in various stages of degeneration of no importance whatever in the causation of the growths. The question must still be regarded as an open one, and we must wait for further investigation.

Section 2.

GENERAL DISEASES.

DISEASES OF THE WHOLE BODY, AND DISEASES
WHICH MAY BE DISTRIBUTED IN SEVERAL
PARTS OF THE BODY.

I.—*DISEASES DEPENDENT ON SPECIFIC POISONS.*

THE INFECTIOUS DISEASES.

PYREXIÆ—FEVERS.

Fever is not a series of symptoms. It is a general disturbance of metabolism which gives rise to several symptoms, the most striking of which is a rise in the temperature of the body. The other symptoms are referred to the alimentary system, giving thirst, loss of appetite, diminished digestive and absorptive powers; to the circulatory system, giving quickened heart, enfeeblement, &c., and quickened pulse; to the respiratory system, giving quicker respiration; to the excretions, diminishing them, so that we get scanty high-coloured urine, &c.; to the nervous system, giving headache, discomfort, excitement, delirium, &c.; and to the general nutritive processes of the body, which are disturbed, causing wasting, loss of weight, and tissue degenerations.

The normal temperature of the body.—As determined by the thermometer placed in the axilla, it is generally considered to be 98·6°. In the mouth it is usually about $\frac{1}{2}^{\circ}$ and in the rectum about 1° higher. In children it is generally $\frac{1}{2}^{\circ}$ higher, and in old people slightly lower, than

in adults. A constant normal temperature does not really exist: it is constantly varying within narrow limits, being generally highest in the late afternoon and lowest in the early morning between 3 and 6 a.m. These diurnal variations rarely amount to more than 1° , exceptionally to 2° . They are constant, so that the same average will be found in every healthy individual at the same hour. Hence a general mean of 98.6 is fairly accurate, at any rate sufficiently so for practical clinical purposes.

Equilibrium of Temperature.—Heat is constantly being produced within the body wherever nutrition and function are going on. Most heat is produced where these chemical changes are most active, as in muscle and gland. The circulating blood distributes this heat all over the body, and the surplus is lost in the skin (over 75 per cent.) and lungs (over 20 per cent.). These two factors, heat production and loss, are variable, and in order that an equilibrium should be maintained, the body must possess a regulating mechanism. It is only highly-developed animals with a complex and highly-endowed nervous system which possess a constant internal temperature (the surface temperature is constantly varying), and it is in this nervous system that the regulating mechanism resides. The cutaneous loss is partly due to radiation of heat from the blood-vessels of the skin, and partly to the evaporation of the insensible sweat. The sweat loss may be greatly increased by the production of a sensible sweat. The respiratory loss is due to the radiation of heat and the evaporation of water from the respiratory passages, very little, if any, coming from the lung. In health, when there is increased production of heat, there is a compensatory increased loss and hence the equilibrium is maintained. The mechanism by which increased loss is brought about is complex. Influences from outside affect the nerve endings in the skin, and the impulses are carried to the vaso constrictor or vaso-dilator centres in the medulla,

causing constriction or dilatation of the cutaneous blood-vessels, as the case may be. Thus cold will cause constriction and heat dilatation. Similarly they reflexly diminish or increase the secretion of sweat. Inside influences, like heat, directly affect the vaso-dilator and sweat centres in the medulla and bring about dilatation and sweat secretion. The peripheral nervous reflex is always active and serves to maintain a constant temperature. The central mechanism is slower and serves to restore the constant temperature when it is disturbed. In the light of certain experimental and clinical observations it is believed that heat production is also under the control of the nervous system, and the controlling centres, called thermogenic, are generally placed on the medial side of the corpus striatum. They control the production of heat in the muscles and glands. These are the laws which govern the temperature in health, and they will be seen to have an important bearing upon fever.

Fever or Pyrexia.—It is slight if about 101° , moderate if about 103° , and severe if higher. If it reaches $107\cdot6$ and upwards, it is called hyperpyrexia. The temperatures compatible with life are generally placed between $93\cdot2$ and $107\cdot6$, but higher temperatures have been recovered from. A pneumonic patient with a temperature of $113\cdot9^{\circ}$ and a malarial one with $114\cdot8^{\circ}$ both recovered. In Teale's case 122° was passed several times, yet recovery followed. Generally speaking, infectious high temperatures are more dangerous than non-infectious ones. Recovery does not usually follow where temperatures of 107° or 108° and upwards have lasted long. Fever generally shows somewhat similar diurnal variations to those in health, being highest in the evening and lowest in the early morning. A typical fever shows a certain course, naturally divisible into three stages.

1. The initial stage, during which the temperature continues to rise, and if this rise be rapid and accompanied by

marked contraction of the blood-vessels of the skin, as in malaria, a sensation of cold is experienced and a shivering fit or rigor is apt to follow. Although the surface temperature falls the internal temperature rises.

2. The fastigium or acme follows, during which the temperature is maintained with occasional variations. It may last for days or even two or three weeks. The above-mentioned diurnal variations always occur of course.

3. The defervescence is sometimes short, when the fever is said to end by crisis, sometimes protracted, when it is said to end by lysis. The temperature returns to normal quickly and suddenly in the former, and gradually in the latter. It is associated with changes in the skin, more favourable to the discharge of heat. The temperature of the skin rises and a more or less abundant secretion of sweat occurs. At other times there is free diuresis or diarrhœa. These critical evacuations serve to free the body from waste products, extractives, and toxins.

Nutrition in Fever.—The nutritional changes are extensive and may be grouped as follows :—

1. Changes in the Urine.—In addition to the ordinary well-known changes affecting amount, concentration, colour, &c., the urea extractives and phosphoric acid are increased and the chlorides usually diminished. The amount of nitrogen taken in by the food is reduced to a minimum, but the amount discharged by the kidneys is greatly increased, far more than it would be in a similar diet in health. Albumen, peptone, albumose or deutero-albumose may also appear.

2. Changes in the Respiration.—The respiratory change is not materially altered, the respiratory quotient $\frac{\text{CO}_2}{\text{O}_2}$ remaining the same.

3. Degenerative Tissue Changes.—The blood loses red blood-cells and becomes deteriorated, the muscles lose

glycogen and weight and degenerate, the fat loses weight, the liver loses glycogen and shows functional troubles, and the secretions, *e.g.*, saliva, gastric, &c., are all diminished. The glandular organs degenerate. The granular and fatty degenerations may follow from mere high temperature, but the others are caused by toxins.

The Production of Fever.—These changes show that destruction of tissue is going on. The increased loss of nitrogen demonstrates the loss of tissue albumen. The body is feeding upon its own organic albumen and fat, the nitrogen being excreted by the kidneys and the carbon by the lungs. This causes increased production of heat. In health, heat production tends rather to exceed than fall short of the needs of the body; in fever it advances still more. There is then in fever both increased production and sometimes also diminished loss of heat. Both these processes are under the control of nerves and nerve centres, and in fever these become affected. Both the nerves and nerve centres become capable of only partially responding to abnormal impressions, and hence are no longer able to efficiently discharge their function. The primary causal toxin is the cause of the disturbance. When it is overcome or eliminated, the increased heat production ceases, the mechanism presiding over loss resumes its full vigour, and the fever ceases.

Causation of Fever.—Any substance capable of influencing the heat producing or losing mechanism may cause fever. They are very numerous, but their action varies with the dose, mode of administration and the subject. They may be classified into inorganic, organic and microbic. The inorganic include the venous injection of large quantities of distilled water and the exhibition of phosphorus. The organic are more numerous, *e.g.*, strychnine, eserine, cocaine, caffeine, &c. Blood or its serum or its fibrin ferment

injected into the body produces fever, and so do the extracts of organs and the urine. The fever of chlorosis is due to the gradual destruction of red blood-cells; of paroxysmal hæmaturia to their rapid destruction. Again, severe fractures or contusions are sometimes followed by fever, caused by the absorption of blood effusion or cell debris, for it is possible that all cells contain a pyrogenetic substance. Again, some of the albuminous products of digestion, such as albumose or deutero-albumose, have great pyrogenetic power; but the most frequent causes of fever are microbic in origin, viz., the poisons produced by microbes. They differ from purely chemical poisons by generally showing an incubation period and by acting in very small doses. Tuberculin has been shown to contain deutero-albumose.

Clinical Types of Fever.—(1) Continuous, where the fever exists for some time at a fixed level; (2) Remittent, when several successive rises are followed by falls which do not reach the normal; (3) Intermittent, is similar but the falls do reach the normal; (4) Relapsing, is but a variety of the last, but the apyrexia periods are longer; (5) Specific, when caused by a specific germ. There are still other varieties of fever, such as hysterical and urethral, which are probably mere hyperthermias and not true fevers, which are always due to a toxin circulating in the blood. These others may, on the other hand, be due to some simple nervous action.

The role of the Increased Temperature and Altered Nutrition in Fever.—The rise of temperature is one of the manifestations of the reaction of the organism to the cause. It is the result of its defensive powers being put in action. If excessive it is hurtful, as all reactions, however good in themselves, may be if carried to excess. Simple hyperthermia if too high or carried to excess is dangerous, and toxic hyperthermia is still more so; but so long as the rise

of temperature is under control and not excessive, it is useful and beneficial. It increases phagocytosis and the bactericidal powers of the serum, and thus favours the destruction of microbes and the elimination of their toxins. The nutrition is modified, not only during the disease but for a long time afterwards. Weak infants often become vigorous after fever, and increased nutrition is seen, especially after typhoid. The nutritional processes may also then be beneficial and the whole feverish process designed to antagonise the cause, and hence good and beneficent.

General Treatment of Fevers.—In the treatment of fever there are certain general indications which are worthy of attention. At the onset of the disease, when the specific character of the fever is as yet uncertain, an emetic or a mild laxative may be given, followed by a diaphoretic mixture. The bowels are thus relieved, and the action of the skin promoted. The feverish symptoms accompanying a catarrhal attack are often alleviated in this way, and not infrequently an apparently severe catarrh may be cut short. In the case of children suffering from a feverish attack, a hot bath at night comforts the little sufferer, and promotes sleep, the child being quite well in the morning. In giving purgatives, the mildest should, in the first instance, be alone used; for should the attack turn out to be one of typhoid fever, much mischief may be done by too free purgation. In the course of an attack of fever much care will be required in the treatment of the complications as they arise. One of the most important of these is hyperpyrexia, or a continued and abnormal rise in temperature. The gravity of this state will depend on the nature of the fever, its probable duration and character. Thus, a temperature persistently high in typhoid, a fever of some duration, is of far greater gravity than a like temperature in relapsing fever. It must also be borne in mind that in some fevers—scarlet fever

and relapsing fever—a high temperature is characteristic of the disease. We have, therefore, to bear in mind the duration and character of the fever before we decide as to our prognosis of its probable result. To regulate the temperature, ice may be applied to the head, chest or abdomen, or the body may be sponged with cold or tepid water; but by far the most important antipyretics are the “wet pack,” cold or tepid, and the cold bath. In the former the bed is first protected by a waterproof sheet, and on this is placed a dry blanket, on this again a blanket wrung out in either cold or hot water is placed, and then the patient is carefully wrapped up in two blankets. In these he may remain for half-an-hour, be then removed, carefully dried, and put back to bed. The efficacy of the cold pack is very greatly increased if iced water be used. The cold bath is a favourite method of treatment with Leibermeister in typhoid fever and has recently again come into vogue. It consists in placing the patient in a bath at a temperature of about 80° F., and then rapidly lowering it by the addition of ice to 60° or 50°. In this the patient may be kept for from fifteen to thirty minutes, but he should be at once removed on the slightest appearance of collapse, and means taken to assist a healthy reaction. In cases of marked and persistent hyperpyrexia, this treatment has undoubtedly saved life. Collapse from excessive shock should always be guarded against by the administration of alcohol or other stimulant when necessary. Quinine, in one large dose, 15 to 30 grs., given at night, has been recommended in the treatment of the hyperpyrexia of typhoid. In typhus its use is doubtful. It is of great importance in the treatment of fever that the services of an experienced fever nurse be obtained, and that the nursing of the patient be entrusted as little as possible to his friends or relations. In the course of fever so many anxious and critical periods occur that it is

never well to leave to relations the duty of watching the sufferer. In the next place, a large and airy room should be provided, and if the house be situated in a street, it is as well to select a back room away from all noise. Some fevers—smallpox, scarlet fever, &c.—may with advantage be treated in tents, or wooden huts, which may be pulled down when the epidemic has passed.

The room selected should be as large and airy as possible, have abundance of light and be situated at the top of the house. All carpets, bed hangings, and useless furniture should be removed, and if possible the room should contain two beds of the same size and height from the floor, so that when one bed is drawn up alongside the other, the patient can be shifted far more easily and safely than would be the case if the patient were moved from one part of a large bed to another. Only those who have been ill for some weeks, or even days, can appreciate the comfort of being moved from a hot and uncomfortable bed into clean sheets. A firm straw palliasse with a good hair mattress upon the top is best; this is generally preferred to a wire mattress owing to its being firmer. In certain fevers a water bed is of the greatest value, and especially where evacuations are passed involuntarily. The position of the bed in the room is of great importance. It should be so placed as to permit of the physician and nurse being able to walk all round it. The bed-clothing should be light and warm, and always kept scrupulously clean. The services of skilled nurses should be secured whenever possible, and a carefully-written report of the temperature, bowels, urine, nourishment, and medicine should always be kept. The bed-pan and urinal, each containing a small quantity of strong antiseptic solution, *e.g.*, carbolic acid, sanitas, chlorinated lime, &c., should always be advised. This obviates the chances of a chill, fatigue, &c., through the exertion of the patient getting out of bed to the night commode or w.c. The nurse is not

required to sleep in the room during the hours allotted to her for rest. She should have a room to herself, and should also be allowed at least two hours out-door exercise and fresh air daily. The sick-room may be ventilated by a fire, and the temperature of the room should always be regulated by a thermometer.

Administration of Medicines, &c.—As it is noted that towards night fevers increase in severity, the pulse rising instead of falling, and the skin, ceasing to act, becoming hot and dry, so it is argued, and with justice, that the afternoon and evening are the times when febrifuge medicines should be administered. The object in view is to increase the discharge of urea, and also to augment, secondarily, the action of the skin. The excretion of urea is abnormally large whilst the feverish symptoms last. The natural tendency is for the excretion of urea to become less towards evening; but, as before said, the formation is increased during the fever, which is greatest towards night, and if the discharge of the urea be prevented, dangerous complications result. Our object, therefore, is to help the discharge of the urea and other waste products and promote the action of the skin. Febrifuge medicines and diluents are therefore indicated. Purgatives do not alone meet the requirements of the case. On the other hand, the time for administering food and alcohol is in the fore part of the day; but when the fever has entirely subsided, during the subsequent debility and convalescence, nourishment should be given in the evening and during the night. Stimulants and tonics are best given morning and evening, and also during the night should the patient awake. The patient may often with advantage be aroused from sleep to have nourishment administered, where there is marked prostration.

The Diet in Fever.—The diet in fever is important. It must always be borne in mind that in a case of fever we

are dealing with a disease that may last from three to four weeks and more. It therefore requires more than ordinary care to regulate the amount of nourishment given at those critical periods just when the recovery or death of the patient may occur. Some years ago it was the custom "to starve a fever," and there can be no doubt that many deaths resulted from this method of treatment, for many of the symptoms which were then held to indicate inflammation of the brain or its membranes, or of the stomach, were in reality the indications of protracted abstinence. Thus, pain in the head, epigastric tenderness, thirst, vomiting, determination of blood to the head, suffusion of the eyes, headache, sleeplessness, and delirium, may be the symptoms of the deprivation of food. Fever patients have a great aversion to take food: it is therefore necessary to insist on its being taken as a medicine. In the early stages, if the patient be strong and robust, barley water and milk, with occasionally some beef-tea or mutton broth, should be ordered, and there is no objection to gruel made with milk. In typhoid fever only slops should be given, and a return to solid food made with the *utmost* caution. As the disease progresses, strong beef-tea, jellies and alcohol must be given, remembering from what has been said above, that a red-glazed tongue and delirium are not contra-indications to the use of the last-mentioned. We may therefore sum up the general treatment as follows:—

1. To moderate, when necessary, the violence of arterial excitement by saline laxatives, rest in bed, and low diet.
2. To diminish heat production.
3. To increase heat loss.
4. To support the powers of the system as soon as they begin to flag.
5. To obviate local inflammations and congestions.
6. To relieve any urgent symptoms as they arise.

SUB-GROUP (α)—ACUTE INFECTIVE DISEASES
OF KNOWN BACTERIOLOGY.

RELAPSING FEVER.

Lat. *Febris residua*. Fr. *Fièvre à rechutes*.

Ger. *Typhus recurrens*.

FAMINE FEVER, SEVEN-DAY FEVER, BILIOUS REMITTENT FEVER,
SPIRILLUM FEVER.

DEFINITION—A continued fever of short duration, characterised by absence of eruption, and an abrupt relapse occurring after an interval of about a week of comparative health succeeding the first attack.

Etiology.—The spirillum or spirochæte, discovered by Obermeier, in 1873, in this disease and in no other, is now recognised as the specific cause. His observations have been fully confirmed, and the disease has been produced both in man and monkeys by inoculations with blood containing the germ. It is a delicate spiral filament of extreme tenuity but of considerable length (two to six times that of the diameter of a red blood-corpuscle). It shows several regular sharp curves, like a corkscrew but abrupter, has finely pointed ends, and is actively motile. It begins to appear in the blood shortly before the onset of the fever, and rapidly increases in numbers, becoming very abundant during the height of the fever. It differs in this respect from other bacterial diseases in the human subject, in which the germs are rarely found in the blood during life. They begin to disappear shortly before the crisis, and in the intervals they are not to be seen in the circulating blood, as they accumulate in the spleen (hence its enlargement during the disease), where they are rapidly destroyed. They do not form spores. The first attack probably established a transient immunity, not long enough to allow of the destruction of all the spirilla, hence their reappearance in the blood. This second attack is always shorter and slighter than the first and increases the

immunity so that it now frequently lasts long enough to allow all the organisms to be killed; if not, it may be followed by another or even more attacks before the disease subsides. The spirillum has not yet been cultivated outside the body, and we are ignorant of the conditions of its saprophytic existence; but we do know that famine and filth favour its attack upon man, hence the name famine fever. It is moderately contagious, and neither age (though it is more common in early life), sex, nor season appear to have any influence.

Symptoms.—The invasion of the fever is sudden: chilliness, followed by severe rigors and pains in the head and limbs, ushers in the attack. The patient takes to his bed at once, not so much from weakness, as in typhus, but because he feels “giddy.” The onset is not, however, generally so sudden as in typhus or pneumonia. The period of incubation usually varies from four to sixteen or more days, but in some cases the disease has broken out immediately after exposure to infection. Many cases have walked considerable distances into hospital. The temperature rises rapidly, reaching 102° to 105° F., or even higher. The pulse is rapid, ranging from 110 to 140, or even more. The face is flushed, and the skin hot and burning and frequently jaundiced. In a small minority of cases a rose-coloured rash, similar to, but individually smaller than in typhoid has been observed. The tongue is generally moist and covered with a yellowish-white fur. There is thirst, nausea, and sickness, and often troublesome, distressing, and persistent vomiting of greenish-yellow matter. Intestinal symptoms are not prominent. Constipation is usual, though diarrhoea may occur. Increase of the splenic dulness can be detected early, and later there is tenderness over it. The liver is generally enlarged and tender, though not so marked as the spleen. The respiration is quickened and there is generally a cough. There is often headache, generally

referred to the occiput and vertex, sometimes delirium, especially if the fever be high, and occasionally convulsions in young people. Pregnant women almost always abort, though often not until a relapse has occurred. After continuing with generally increasing severity for five, six, seven, or nine days, the crisis suddenly occurs, generally at night. The temperature falls in a few hours to subnormal, and the pulse to about half its rapidity. There may be a copious perspiration, or diarrhœa, or epistaxis, or various combinations of these phenomena. Convalescence is rapid, and the patient soon gets up feeling quite well, only, however, to be again abruptly struck down after an interval of a week, generally on the fourteenth day from the commencement of the first attack, with a recurrence of the same symptoms. The relapse is usually shorter, though often more severe than the first attack, lasting often three, sometimes four or five days, and is followed by another period of convalescence, which is ushered in by a crisis nearly as abrupt as the first. This convalescence is frequently permanent, though a third or fourth, or even fifth, attack may occur.

Prognosis.—Relapsing fever is seldom fatal. Complications are not common. Pneumonia is most often seen. Parotitis, nephritis, and hæmaturia have been met with. Recently it has been shown that when the spirilla are rendered motionless within an hour by the blood of another patient who has passed through one paroxysm and is expecting another, no second paroxysm will occur, while if they are not quieted within two hours another paroxysm is sure to follow.

Diagnosis.—An early diagnosis can be made only in one way, viz., by a microscopic examination of the blood. A drop of blood shows the spirilla in vast numbers moving quickly across every field of the microscope and

disturbing the blood-corpuscles. A cover glass film of the blood may be dried, and stained with Löffler's methylene blue. Later, when the typical course of the fever has shown itself—its sudden onset, definite duration, and sudden disappearance, followed after an interval by sudden reappearance—the diagnosis can be made clinically also with certainty.

Treatment.—No drug has yet been discovered which affords much relief in this fever. Quinine, from which we should expect much, is almost useless, and we must confine our attention to treating the leading symptoms, and by supporting the patient's strength allow the disease to wear itself out. For vomiting, often very distressing, bismuth and hydrocyanic acid and effervescing alkaline drinks (potash and soda water) should be tried, and the administration of repeated small doses of ipecacuanha wine (2–5m) is frequently beneficial. A mustard leaf over the stomach is well worth a trial where all else fails. Opium is the only other drug of really great value, as it relieves the awful headache and muscular and joint pains. Frequent sponging of the skin with tepid water or water and vinegar is a source of great comfort to the sufferer. Other symptoms, when they arise, must be treated as the occasion requires. Antipyrine and antifebrin in small and repeated doses may be found useful. As this fever is undoubtedly contagious, strict isolation is requisite.

Geographical Distribution.—Relapsing fever is indigenous to Ireland. Epidemics have occurred in the British Isles, and also in Poland, Germany, and Russia. It has on various occasions been introduced into America, but has not spread in that country. It has been noticed occasionally in India, in Egypt, and Algeria. It has probably been confounded with tropical-yellow fever, or bilious remittent fever of malarious origin.

TABLE showing Points of Distinction between Relapsing, Typhus, and Typhoid Fevers.

	RELAPSING FEVER.	TYPHUS.	TYPHOID.
<i>Duration</i> ..	Seven-day fever. Relapses occur on the 14th day from the invasion; the 7th from the critical sweating.	Fourteen-day fever.	Twenty-one day fever.
<i>Mortality</i> ..	About 4%.	10-12%.	7-20%.
<i>Age</i> ..	All ages.	Adult Life.	Youth and adolescence.
<i>Tendency to occur</i>	Epidemically.	Epidemically.	Endemically.
<i>Anatomical Signs</i> ..	Characteristic eruption absent. Petechiæ present, differing from typhus by not appearing in the centre of exanthematous spots	Mulberry rash. Petechiæ present and persistent.	Rose coloured rash, occurring in successive crops; congestion, and ulceration of Peyer's patches.
<i>Diagnosis</i> ..	Muscular pains, Tenderness and enlargement of liver. Sudden cessation of fever. Delirium rare. Discovery of the spirilla in blood.	Active delirium frequent. Peculiar dusky coloration of skin.	Roseola present. Diarrhoea present. Low muttering delirium. Vidal's reaction.
<i>Cause</i> ..	Starvation and over-crowding predispose. The spirillum obermeieri is the exciting cause.	Over-crowding and starvation predispose. Specific germ not yet known.	The typhoid bacillus.

TYPHOID AND TYPHUS

(INTRODUCTION).

Before entering upon a discussion concerning the nature of two of the most important continued fevers, *Typhus* and *Typhoid*, it may be as well to place in parallel columns a list of the principal points in their symptomatology and pathology, which may help to distinguish them, particularly as the bacteriological classification adopted places them apart in this edition:—

TYPHUS

attacks persons of both sexes and all ages indiscriminately, and is generally traceable to contagion.

The invasion is sudden; the head symptoms in the course of the fever are severe, delirium with contracted pupils usually occurring towards the end of the first week. The eruption, which belongs to the class *maculæ*, appears on the skin in the form of a mulberry-coloured rash, between the third and seventh day of the disease, imperfectly disappearing on the application of pressure, and continuing until the termination of the fever. The rash is seen all over the body, but seldom appears on the face. The spots may become petechial. Diarrhoea is seldom present during the course of typhus, and hæmorrhage from the bowels never occurs.

TYPHOID

chiefly occurs in the young, from twelve to twenty-five years of age, and but rarely after forty. It is during this period that the solitary and aggregate glands of Peyer are most fully developed. As age advances, these glands atrophy, so that after forty-five only traces of their previous existence can be discovered. The lesion in these glands forms the "anatomical sign" of typhoid.

The invasion is insidious, the premonitory symptoms lasting about a week. The abdominal organs are much affected; congestion and ulceration of Peyer's patches occur, with attendant diarrhoea; and there is frequently more or less hæmorrhage from the bowels. The abdomen is tense, and pain is felt on pressure. There is low muttering delirium, with dilated pupils. The eruption, which belongs to the class *papulæ*, appears about the seventh day, and is chiefly found on the chest and abdomen. It comes out in successive crops of rose-coloured, slightly-elevated lenticular spots, in size somewhat less than a split pea. The rash disappears on pressure. In two or three days the spots fade away, to appear again on another part of the body. "This successive daily eruption of a few small very slightly-elevated rose-coloured spots, disappearing on pressure, each spot continuing visible for three or four days only, is peculiar to, and absolutely diagnostic of, typhoid fever."

TYPHUS—*continued.*

Typhus prevails most frequently among the poor, and is a disease of adult life. It is thought to be due to bad diet and over-crowding. The disease runs its course in from fourteen to twenty-one days, and is most dangerous about the end of the second week.

Typhus fever is most frequently fatal before the fifteenth day, never after the twentieth. The tendency to death is by coma.

TYPHOID—*continued.*

The average age of mortality appears to be about twenty-three. The rich are most frequently attacked. It is caused by a specific bacillus (Eberth's). The pollution of drinking water by sewage is the most frequent source of infection. Typhoid is rare in pregnant women.

Relapses are not uncommon in typhoid. The duration of the fever is more or less prolonged, from three to six weeks, but its maximum of intensity is reached about the third week. Typhoid is more frequently fatal after the twentieth day; more than half the deaths occur after this period. The tendency to death is by asthenia.

TYPHOID FEVER.

Lat. *Febris Enterica*. Fr. *Fièvre Typhoïde*. Ger. *Abdominal-Typhus*.
Syn. *Ileo-Typhus*.

ENTERIC TYPHUS—PYTHOGENIC—ABDOMINAL TYPHUS.

DEFINITION—A continued fever, characterised by the presence of rose-coloured spots, appearing in successive crops, chiefly on the abdomen, with a tendency to diarrhoea, with specific lesion of the agminated and solitary glands of the intestines. The period of incubation is from six to twenty-four days or even longer. Enteric fever occurring in the child is often named infantile remittent fever.

Typhoid most frequently attacks the young under the age of twenty-five. After that age it rapidly and uniformly diminishes in frequency. One attack of typhoid is believed to confer immunity from subsequent attacks, as in the other infectious eruptive fevers. The onset of the disease is, as a rule, gradual and insidious. In rare cases, however, the invasion is as sudden and pronounced as in typhus. I have seen a case at first mistaken for a severe gastric bilious attack.

Etiology.—Typhoid fever occurs most frequently in the months of October, November, September, and August, its prevalence being increased by continued excessive heat, and diminished by prolonged cold. In dry regions and seasons the poison is probably disseminated to a considerable extent by dust. The experiences in many places during the late South African War seem to support this. Typhoid fever appears to arise from a special poison contained in the excreta of infected persons. It is believed to be due to the bacillus of Eberth—a short, thick, highly motile bacillus with rounded ends. It usually enters the body by *the drinking water*, but it may enter with the *food*. Milk is the food most frequently contaminated, but ice, salads, and oysters may carry the infection. Flies may carry the infection to food, especially in an epidemic or in camp. The bacilli may live for weeks in water (particularly when it is comparatively pure), in which they probably multiply. Milk is a suitable medium, and their growth and multiplication produce no change in the milk. They grow and live in the soil for months. They penetrate the epithelial lining of the intestine, and reaching the lymphoid tissue cause swelling and necrosis. They are found in the lymphoid tissue of the intestine, in the mesenteric glands, in the spleen, in the liver, &c., and also in the urine.

Pathology, &c.—The anatomical characters peculiar to typhoid, and found after death, are seated in Peyer's patches and in the solitary glands of the small and large intestines. The glands forming Peyer's patches are most numerous close to the ileo-cæcal valve on the side of the intestine opposite to the attachment of the mesentery, and those nearest the valve suffer most from the poison of typhoid fever. The glands are found enlarged, congested, and studded with ulcers varying in size.

Changes found in the Glands—

1. Increased vascularity.
2. Proliferation of the lymphatic contents.
3. In colour they are greyish-white or reddish.
4. Contents cellular, soft, and brain-like.

Terminations—

- (a) Resolution. (b) Ulceration. (c) Cicatrisation.

Ulceration—

- (a) One large ulcer, size of gland, or more usually several small ones.
- (b) Edges formed by inflamed mucous membrane, ragged and undermined.
- (c) Base smooth, formed by one of the coats of intestine.

It may be as well here to give the signs which distinguish the typhoid intestinal ulcer from the tubercular:—

TYPHOID ULCER.

— —

1. Long diameter, usually parallel to long axis of the intestine.
2. Edges thin, ragged, and undermined.
3. Does not lead to stricture of the intestines.
4. As a rule heals readily.
5. Seldom accompanied with local or general peritonitis
6. Floor smooth
7. Perforation common.
8. Limited to Peyer's patches and solitary glands.

TUBERCULAR ULCER.

— —

1. Long diameter, usually transverse to long axis of the intestine.
2. Edges thick, undulated, indurated, not undermined.
3. May lead to stricture.
4. Seldom heals.
5. As a rule accompanied with local or general peritonitis.
6. Floor rough and nodular.
7. Perforation rare.
8. Not so strictly limited to these areas

Other lesions are also present; the spleen is more or less enlarged and softened, the walls of the heart flaccid, and the muscular tissue softened.

The blood shows no leucocytosis. The red cells and hæmoglobin fall considerably by the third week and there

may be great post-typhoid anæmia. The kidneys are sometimes greatly congested and enlarged, and the uriniferous tubes filled with exfoliated epithelium; at other times they are unusually pale. The lungs are gorged with blood of a dark colour, and present a spotted appearance, both externally and internally (*Wilks*). Consolidation is found at the posterior edge of the lung, seldom passing beyond the stage of red hepatization. The pneumonia is lobular, patches of the lung being attacked. The mesenteric glands are also enlarged and softened. The membranes of the brain may or may not be inflamed and congested, the delirium being no index of the intra-cranial disturbance.

Symptoms.—During the premonitory stage of typhoid fever, the patient is chilly, indisposed to exertion, and hangs over the fire. His limbs tremble, he complains of feeling sick, with entire loss of appetite. Pains in the limbs, of a rheumatic nature, are complained of, and he may suffer from repeated attacks of hæmorrhage from the nose. His breath becomes offensive, his throat sore, his bowels usually irritable, sometimes confined, his sleep broken, his pulse quickened, his tongue white; he daily becomes weaker, and at last may have a violent rigor (rare), followed by great heat of the skin and intense headache, and such muscular debility that he takes to his bed. This is the accession, and the course of the fever has now set in, and may be divided, for the sake of convenience, into three weekly periods:—

First Week.—In the first week, the leading characters are vascular excitement and nervous depression. The fever during this period generally shows a gradual and steady increase, the evening temperature being about a degree-and-a-half higher each day, and reaching 103° , 104° , or 105° .

There is a frequent and bounding pulse, often dicrotic, of from 90 to 110 or more, keeping pace more or less with the rise in temperature. The pulse, however, in the

presence of marked fever may fall to 60 or 50 per minute, and it has even been known to fall as low as 37. The respirations are also increased and are frequently accompanied with a slight cough. There is great thirst, and at night there is great restlessness, confusion, and even delirium if the fever be high. Sudden sweats are not infrequent.

The temperature during the latter part of the first week and the early part of the second is almost uniform. The maximum is reached on or about the fourth or fifth day, and then remains stationary with slight morning remissions till about the end of the second week, decided defervescence not usually taking place till the twenty-first day, though in many cases it is as early as the fourteenth.

During the third week the temperature usually becomes more markedly remittent, the differences between morning and evening temperatures generally exceeding 2° . In typhus the normal temperature is regained about the fourteenth day; in typhoid, the fever is continued with various intermissions into the third week, falling by lysis until it reaches normal in the evening. This generally occurs about the twenty-first day. Variations in this typical temperature course are common. The rise may be sudden and not step like, and so may be the defervescence. A sudden considerable fall may follow an intestinal hæmorrhage. When the temperature for two successive evenings has closely approached to normal, convalescence has set in.

The sleep is broken by frightful dreams, the patient often muttering, or uttering in a loud voice, portions of detached sentences. When awake, he lies with his eyes half open, quite conscious, but showing little interest in what is going on around him. In the course of the delirium, the patient may be temporarily restored to consciousness by the entrance of a stranger into his room, or by a sharp question addressed to him.

The oppression of the mental faculties is peculiar, and the patient can only with difficulty be induced to give

an account of himself, or indeed to complain of anything but his head.

The tongue presents no distinctive characters. It is at first slightly coated with a white fur, but appears moist and broad, and is marked by the teeth. The epithelial covering generally falls off, leaving "a moist, red, smooth tongue, that looks as if covered with gold-beater's skin, or else is already inclined to dryness."

The bowels may be confined at the outset of the fever, or even remain so during the entire attack, with an occasional loose motion; but towards the end of the first or during the second week, the characteristic diarrhœa often sets in, consisting of light-coloured, semi-fluid, yeast-like or pea-soup stools, of which the patient may have, perhaps, seven or eight or more in the twenty-four hours. The stools may become very offensive. The bacilli are not found in the stools till after the end of the first week and their isolation therefrom is hopeless after the tenth day. The abdomen feels full and tense, and there is slight tenderness felt on deep pressure, especially when made over the right iliac region. A gurgling sensation—ileo-cæcal gurgling—is also experienced by the fingers. Deep pressure is not without risk and as these signs are unimportant, it should not be practised.

The spleen becomes enlarged towards the end of the first week, being sometimes two or three times its natural size, due to increased vascularity and the production of lymphatic elements, but owing to its softness, and to its displacement backwards by the distended intestines, its outline can only be defined with difficulty.

Emaciation rapidly sets in, the muscles wasting as well as the fat. The urine is scanty, high-coloured, and has the ordinary febrile characters: there is often retention at first. During this week the solitary and agminated glands—Peyer's patches—of the intestine become swollen and infiltrated by the proliferation of the lymphatic contents.

They are first reddened, but in a few days become grey, fawn-coloured, and even white. The microscope reveals an early hyperæmia, followed by a large formation of new cells, with large nuclei, having all the characters of lymphocytes. The glands may rise above the surface of the mucous membrane two, three, or more lines in height, their surface being sometimes convoluted.

Second Week.—During the second week, the fever is continuous. The rose-coloured eruption now makes its appearance in successive crops, and is chiefly found on the chest and the front and sides of the abdomen; it generally appears between the seventh and the tenth days, and each crop lasts three or four days. It has generally disappeared by the middle of the third week. If the finger be passed *very* lightly over the surface of the body, the eruption can be detected as slightly-elevated soft pimples, unlike the hard “shotty” feel presented by the pimples in the early stage of the small-pox eruption. The eruption disappears on pressure, and when it fades away no scar is left. The eruption so pathognomonic of typhoid will often have to be carefully sought for. In many cases the spots may scarcely exceed a dozen in number, and in children they are not infrequently absent. Sometimes a scarlatinoid eruption precedes the characteristic eruption of typhoid. The liability to *sudden relapses*, attended with the appearance of new crops of the eruption, is very characteristic of typhoid fever. The headache becomes less, the pallor of the face greater. Increasing emaciation and weakness, a greater tendency to lie on the back, a listless stupor, the voice weaker, and the speech often unintelligible, together with calm or furious delirium, and coma vigil, mark the second period. The tendency to death during the second week is from coma, and then without any marked brain lesions. Towards the close of this period the tongue becomes dry, in severe cases very dry, cracked and covered with a brown fur. Sordes form

on the teeth. The dejections are foetid and often passed involuntarily, sometimes containing blood. The abdomen also becomes distended with gas, so marked in fact as to constitute a condition known as "meteorism"—a very grave symptom if severe, but not so if moderate. There may be a slight cough, sibilant râles, and some amount of hypostatic pneumonia. The fever is still continuous, the temperature vacillating between 102.2° and 104° F. During the second week the swelling and infiltration of the glands continues and reaches its height from the eighth to the tenth day. Resolution may now occur and the disease subside, but if the swelling be great, anæmic necrosis sets in. Bile-stained sloughs form and separate, leaving ulcers. Bacilli are present in groups in the swollen tissue previous to ulceration. The necrosis is mainly due to their action. They are scanty in the floor of the ulcer. These local changes are generally most severe near the ileo-cæcal valve. The mesenteric glands are swollen.

Third Week.—During the third week the fever becomes more markedly intermittent, and the symptoms either slowly decrease in severity or are increased: in the latter case, the *typhoid state*, the emaciation and debility become extreme. The patient lies extended on his back, sinking towards the foot of the bed, making no effort to change or preserve his position. The pulse is quickened and irregular, the respiration more difficult, and severe and debilitating sweats now occur, accompanied with sudamina. There is a purplish flush on his cheeks, and sordes cover his gums and lips. His tongue is dry, shrunken, stiff and black, like a bit of leather; his urine, from inaction of the bladder, is retained, and his fæces pass involuntarily. An alarming symptom at this stage is to find the patient lying helpless at the lower part of the bed.

The noisy delirium, more common in typhoid than in typhus fever, now gives place to increasing stupor; he no longer recognises his friends, but mutters incoherently, or

picks vacantly at the bed-clothes, and if he recovers, he will not remember anything that has occurred during his illness. An erythematous rash in most cases appears on the sacrum, the cutis becomes exposed, and bed-sores supervene. At length, and sometimes suddenly, as if by a crisis, he begins to improve; the tongue becomes moist at the tip and edges; there is some return of reason, like that of a little child; he asks for food in a ravenous way, and expresses a desire to get up. The intestinal ulceration characteristic of this period generally involves part of the circular coat of the bowel, but it is very variable, both in extent and depth. The edges of the ulcer are usually soft and undermined and the floor smooth.

Still, however, there is a stage of sequelæ, in which he suffers from internal mischiefs which may have already taken place, and from one of which he may even yet be carried off. The distinction so often drawn between sequelæ and complications is unnecessary and is not scientifically accurate. Among these sequelæ or complications may be mentioned sudden heart failure about the third or fourth week, peritonitis from perforation of the intestines, bronchitis, pleurisy, pneumonia, erysipelas, mumps, hæmorrhage from the bowels, and ulceration of the larynx just below the vocal cords. Wilks describes a case in which the ulcer in the larynx "led into a space between the trachea and œsophagus, through which the air had escaped, and had given rise to general emphysema." A complication like this may give rise to the emphysema that has been sometimes noticed during the course of typhoid fever. It is at the end of the third week that death most frequently occurs. The bowel affection may itself prove fatal, either in an early stage by hæmorrhage (3 to 5 per cent.), or in the convalescent stage, about the fifth or sixth week, by perforation of the bowel and escape of its contents causing peritonitis. Perforation may occur either during separation of the slough or by an extension of the ulceration. This is to be feared when, after an

attempt at recovery, the patient still suffers from irritable bowels, with occasional hæmorrhage, the tongue remaining preternaturally red at the tip and edges and the pulse frequent. When perforation takes place, the abdomen suddenly swells and becomes excessively tender, the patient generally dying in three or four days. Exclusive of the sequelæ this is a brief account of a typical case of typhoid fever, but it must be borne in mind that the disease is liable to marked variations: thus the attack may be so slight as to be unrecognised, the patient going about his usual work. To these cases the late Dr Fagge applied the term *ambulatory typhoid*. Hæmorrhage or perforation may be the first symptom.

In some other cases peritonitis may cause death, the result of an undetected typhoid fever. Some attacks may even abort before the third week, and the recognition of these cases is of importance as they may become the foci of an epidemic, having been mistaken for simple febricular or gastric or intestinal catarrh.

Diagnosis.—A careful consideration of the symptoms and course will distinguish it from typhus. In the early stages of mild cases the disease may be mistaken for a somewhat severe form of gastric disturbance with dyspepsia. The early prominence of symptoms relating to special organs, *e.g.*, meningitis, pneumonia, &c., may mislead.

It is said that typhoid may be excluded under the following conditions of temperature, but this is doubtful—

1. If the temperatures on the evenings of the second, third, or fourth days are only approximately normal.
2. If the temperature on the first three evenings be the same.
3. If the temperature on two of the first three mornings be the same.
4. If the temperature on the first two days rises to 104°F.

The absence of leucocytosis is an important guide, but the reaction known as WIDAL'S TEST is by far the most important practical aid to diagnosis. A little blood is taken from the finger or ear of the patient and its serum is carefully diluted at least fifty times. A young culture (less than twenty-four hours old) of the typhoid bacillus on agar is added to a drop of sterile water. The bacilli should move actively and freely. A little of the diluted blood serum is now added, and if the case be typhoid, the bacilli will be presently seen to move about less actively, and then gather together in clumps; whereas if the case be not one of typhoid, the bacilli will continue to move freely and will show no clumping. An hour or so should be allowed, and if agglutination has taken place in that time it is a case of typhoid, if not the test should be repeated. Successive negative results practically justify the exclusion of typhoid. Accurate dilution of the blood-serum is essential and a less dilution than 1 in 50 is hardly safe. The reaction occurs in about 95 per cent. of all cases of typhoid, and those cases in which it fails are generally severe and clinically unmistakable. So far as is known, the reaction, which must always be *complete*, is not given with the blood taken from any other disease. The blood is preferably taken after the sixth or seventh day of the disease. If a negative result be given at first, the test should be repeated at a later stage, once or oftener, as the clumping power of the blood-serum is sometimes late in appearing. A similar reaction can be used as a means of diagnosing cholera, plague, glanders, &c., by using a culture of the specific organism in each case.

Other bacteriological aids are (1) the finding of typhoid bacilli in the urine (this is possible in fully a quarter of all cases); (2) finding them in the blood of the rose spots; and (3) cultivating them from the general circulating blood. It has been recently claimed that this may be done in over three-fourths of all cases.

It may be especially valuable when the Widal reaction is delayed.

Prognosis.—The prognosis will depend upon the duration of the fever, the absolute height of the temperature, the condition of the pulse, the presence or absence of intestinal hæmorrhage, perforation of the bowels with peritonitis, and pneumonia or bronchitis. Patients when not dying from perforation of the intestines may die from paralysis of the heart or brain, the result of long-continued high temperature. The greater the daily fluctuations the better, and the presence of even well-marked remission of the pyrexia adds greatly to a favourable prognosis. A strong, not too frequent, pulse betokens no immediate danger, and, other things being equal, is decidedly in favour of the patient. Constant and prolonged cerebral disturbances are signs of the utmost gravity.

Treatment.—There is no agent yet discovered which can destroy the typhoid bacilli or counteract the toxins produced by them. The most important part of the treatment of typhoid fever depends upon hygienic and supporting measures. The patient should be placed in a large, well-ventilated room, from which all unnecessary articles of furniture have been removed. Strict orders should be given that the bed pan is always to be used, and the stools and even the urine should be disinfected with carbolic acid solution, 1 in 20. On no account should the patient be allowed to get up out of bed until all danger of perforation is past. If seen during an early stage of the disease, a hot bath will often afford great comfort to the patient. The use of the cold bath is strongly recommended by many German physicians, and is certainly of use when the temperature is persistently high and prolonged. In severe cases Leibermeister recommends that the baths should be repeated every two hours, night and day if necessary, so that twelve baths be given in the

twenty-four hours, and in some instances more than two hundred baths have been administered during an attack of the fever. Any temperature in the rectum above 104.5° F. indicates the necessity for a bath. The method adopted has been before described (page 48).

The diet should consist of milk, custard, beef-tea, chicken-jelly, soups, and light-boiled eggs. Alcoholic stimulants are indicated if the pulse becomes feeble, small, and compressible, but in the early stages they are not required. Calomel has been recommended in the treatment of this fever. "I have given this medicine, with but few exceptions, to every case of the fever admitted before the ninth day of the disease. I usually give three or four eight-grain doses during the first twenty-four hours. After having employed this method now on about 800 patients, I still feel that I have every reason to continue it and to recommend it to others" (*Leibermeister*). The use of intestinal "antiseptics" is much to be recommended: calomel acts in this way, or perchloride of mercury in $\frac{1}{32}$ – $\frac{1}{16}$ grain doses (*Broadbent*); but these drugs are only to be given during the first few days of the fever. It is safer to give salol, 10–20 grains, thrice daily during the later stages of the fever. When the temperature remains persistently high, *Leibermeister* recommends the use of quinin in doses so large that from twenty-two to forty-five grains should be taken "*within the space of half-an-hour or, at most, an hour.*" This dose is not repeated for twenty-four hours, in most cases not for two days. Quinin is rapidly excreted by the kidneys, so that to produce its antipyretic action it must be given in large and frequent doses, long intervals in its administration lessening its value for this purpose. Phenacetin in 10–20 grain doses is an admirable antipyretic, and digitalis may be given, but care is required, and the more frequent the pulse the greater the caution necessary. A mixture containing chlorate of potash and syrup, acidulated with hydrochloric

acid, may be given, and was the chief medicine employed during a severe epidemic of typhoid, and with the best results. The diarrhœa, if excessive, may be checked by the administration of the chalk mixture, or lime water. In any case if the bowels are moved more than twice in twenty-four hours, attended with great prostration, astringents should be given—the mineral acids to be preferred. Never be in a hurry to give aperients because patients wish it. As both typhus and typhoid run a definite limited course, the object of the physician is to keep the patient alive till the disease has worn itself out. The bladder should be examined daily, and a catheter used if necessary. In the convalescence of typhoid fever be very careful that solid food be not taken too soon; remember the sensitive, often ulcerated condition of the intestinal mucous membrane, and the liability to perforation.

Treatment of Special Symptoms in Typhoid.

Diarrhœa.—Care should be taken in checking the diarrhœa of typhoid, and unless the patient passes more than four stools daily it should be left alone. If, however, the diarrhœa be severe, lead acetate, gr. 2, diluted acetic acid, 15m, acetate of morphia, gr. $\frac{1}{6}$, may be given. Bismuth with Dover's powder or with small doses of opium may also be tried. A starch enema with 30m of laudanum or opium suppositories are often efficacious. Arrowroot, the only starchy food admissible, may be given boiled in milk; and beef tea, chicken soup and mutton broth must be cautiously administered. In some cases beef-tea, soups, &c., may have to be suspended for a time, and boiled milk substituted.

Hæmorrhage from the bowel is best treated by full doses of acetate of lead combined with opium or turpentine in 10m capsules. Restrict the food and give ice freely to suck. Cold compresses to the abdomen and cold injections into the bowels have been recommended by

some authorities. Hypodermic injection of brandy, ether and strychnia may be necessary. Absolute rest in the recumbent posture must be insisted on.

Tympanites and Abdominal Pain are best treated by hot or cold (ice) applications to the abdomen or by hot turpentine stupes, and covering with a roller bandage of soft flannel. When the gas in tympanites is situated chiefly in the colon an enema of turpentine is of service, and internally salol, β -naphthol, bismuth salicylate and wood charcoal may be employed to relieve distention of either large or small bowel. Where paralysis of the gut has occurred or distention is extreme, the introduction of a long flexible tube into the bowel often gives temporary relief.

Peritonitis is best treated by hot applications to the abdomen—turpentine stupes, linseed meal poultices, &c.—and by the hypodermic injection of morphin. If perforation has taken place, laparotomy is necessary.

Heart Failure must be treated by the free administration of alcohol or ether, the careful administration of digitalis, and the hypodermic injection of strychnia if necessary.

*Bedsore*s must always be carefully looked for and obviated, as far as possible, by careful nursing and attention to cleanliness, the prompt removal of soiled sheets or night dress, and the prevention of constant pressure by placing the patient on a water bed.

Headache is sometimes severe, especially in the early stage of the disease, and much benefit may be obtained from the external application of cold to the head, in the form of an ice cap, and internally by phenacetin given in 10 gr. doses every two hours. It is of great advantage to keep the patient absolutely quiet, and the room darkened.

Insomnia is sometimes a very troublesome symptom and may arise in the earliest stage of the disease. Sulphonal in doses of 15–30 grs. may be employed with advantage. Chloralamide, 15–30 grs., and paraldehyde, 30–60m, are worthy of a trial.

Delirium.—When delirium occurs its cause should be looked for and treated, when possible. It is important to keep in mind that delirium may be the early indication of meningitis, pneumonia, renal trouble, or even intestinal hæmorrhage. Ice bags applied to the head are invaluable for this condition.

Hyperpyrexia must be promptly treated with the cold bath or iced sheet, and an ice bag may be applied to the head. Large doses of quinin have frequently been given with benefit.

INFANTILE REMITTENT FEVER.

Lat. *Febris infantium remittens*. Fr. *Fièvre rémittente des enfants*.
Ger. *Abdominal-typhus im kindesalter*.

DEFINITION—As Typhoid.

The true pathology of this disease was long unknown, till the researches of Dr Charles West proved its identity with typhoid fever. It received its name from the marked remissions in the temperature of typhoid affecting children. It shows greater fluctuations than in adults, but is otherwise the same, and the following separate description is hardly necessary.

Symptoms.—The disease may occur in two forms, a mild and a severe; in the former the symptoms come on gradually and imperceptibly; in the latter the invasion is sudden and the severity marked from the first. The child in the milder form appears ailing for some days, then there is loss of appetite and troublesome thirst; in the daytime he is listless and fretful, and towards evening drowsy and feverish, and the night is passed in a restless and uncomfortable slumber. Sometimes there are profuse sweats, alternating with dryness of the skin, but no relief is afforded to the child by their recurrence. The bowels at first may be

confined and then relaxed, or the relaxation may be contemporaneous with the accession of the disease. The stools are unhealthy, very offensive, and of a peculiar yellow-ochrey colour, and separate on standing into a supernatant fluid and a flaky sediment. The tongue is red, dry, and coated in the middle with a whitish fur, through which the enlarged papillæ are to be seen projecting. The belly is soft and slightly painful when pressed. Sometimes when the child is visited in the morning, it is found sitting up trying to play with its toys, but indifferently pleased at best and requiring constant change. Towards evening all the feverish symptoms return, and these remissions may occur for several days. The pulse is quick, and there is slight cough. An initial bronchial catarrh is even more common than in adults. The above constitute the symptoms of the accession, and to the end of the first week.

During the second week the nights are more restless, the eyes during sleep are half open, the thirst is greater, and some amount of delirium is often present. Sometimes, also, during this period, an exacerbation of the fever occurs about eleven o'clock in the morning, but as the disease abates, the morning attacks cease to recur. The rose-coloured spots, so characteristic of typhoid fever, if present at all, make their appearance towards the end of the second week.

During the third week the severity of the symptoms abates, or, becoming more severe, the case ends fatally. As a rule, however, the severer form of the disease gives some indication of its severity from the very first. The temperature is then high, sometimes reaching 105°F.

Dr West says that, in his experience, typhoid fever in children seldom terminates fatally, and death but rarely follows from those complications so fatal to adults.

Diagnosis.—Gastric catarrh and tubercular meningitis are most likely to be confused with this disease. The

former is distinguished by the more irregular temperature, by the commonness of constipation, and by the character of the stools, while the latter is distinguished by a lower temperature, frequent vomiting, a retracted abdomen, a squint or other cranial signs.

Treatment.—The treatment is to a great extent the same as in typhoid in the adult, a simple saline draught—the citrate or chlorate of potash in syrup and water. If aperients are necessary, senna or castor oil may be given. The diarrhœa may be checked by chalk mixture or hydrargyrum cum cretâ and Dover's powder. A nourishing diet should be given—milk, beef-tea and mutton broth, and wine if necessary. Beef-tea in some cases increases the diarrhœa; when this occurs a change to chicken or veal broth has been most advantageous.

TABLE GIVEN AS AN AID TO DIAGNOSIS.

INFANTILE REMITTENT FEVER OR TYPHOID.	ACUTE HYDROCEPHALUS.	PNEUMONIA.
1. Rare before five years of age, and seldom met with under two.	1. Most frequent under five years of age.	1. May attack children at any age.
2. Vomiting most frequently absent even at the commencement of the fever; absence of nausea.	2. Vomiting most frequently present; constant nausea.	2. Vomiting slight, and soon ceasing; no nausea.
3. Bowels relaxed even at the beginning of the attack; evacuations watery; faces of a lightish yellow.	3. Evacuations scanty, dark, or mud-coloured; deficient in bile.	3. Evacuations natural, but in exceptional cases there may be diarrhoea.
4. Tenderness over the abdomen, greatest in the iliac regions; flatulence well marked.	4. Absence of abdominal tenderness.	4. Tenderness over the abdomen misleading, for children may mean the chest when they say the belly.
5. Tongue with a thin covering of yellow fur in the centre and root, tips and edges red and dry from almost the beginning of the fever.	5. Tongue moist and coated with a white fur in the centre.	5. Tongue redder than in hydrocephalus.
6. Distaste for food, but constant demand for drink, especially cold water.	6. Distaste for drink and food.	6. There is often great thirst.
7. Skin very hot and pungent.	7. Dryness of the skin, but not much heat.	7. Skin hot and dry.
8. Seldom complains of its head; delirium is of early occurrence; hydrocephalus may, however, supervene.	8. Child complains incessantly of its head, true delirium seldom occurring till near the fatal termination.	8. Some slight pain in the head.
9. Remissions—better in the morning, worse at night.	9. Symptoms fluctuate, but no <i>definite</i> periods at which the symptoms invariably remit, or are increased in severity.	9. No remissions.
10. Absence of signs of pneumonia on auscultation till the fever has continued for some time, and then only as a complication.	10. Absence of chest complications.	10. Auscultatory signs of pneumonia.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

Lat. *Febris cerebro-spinalis*. Fr. *Fièvre cérébro-spinale épidémique*.

Ger. *Epidemische meningitis genickkrampf*.

Syn. Malignant Purpuric Fever—Epidemic Cerebro-Spinal Meningitis. Epidemic Meningitis.

DEFINITION—An infectious malignant epidemic fever, attended with painful contraction of the muscles of the neck and retraction of the head. In certain epidemics it is frequently accompanied with a profuse purpuric eruption, and occasionally by secondary effusions into certain joints. Lesions of the brain and spinal cord and their membranes are found on dissection.

Inflammation of the membranes of the brain and spinal column sometimes appears as an epidemic. The first recorded epidemic of this disease occurred at Geneva in 1805. Since then it has appeared in the United States and also in nearly all the countries of Europe but especially in one part or other of Germany. In Ireland its presence has been well marked, but Scotland and England have been less frequently visited.

Pathology.—There is always some congestion and inflammation of the brain and spinal cord found after death, except in malignant cases. The appearances have little to distinguish them from those found in ordinary sporadic meningitis. The meningitic changes may be general or confined to the base of brain and the posterior surface of the cord. The micrococcus intracellularis of Weichselbaum has been found in many cases, and the diplococcus pneumoniæ in others. A similar organism to that of Weichselbaum has been found by Stillé in posterior basal meningitis of infants—possibly a chronic sporadic form of epidemic cerebro-spinal meningitis. The organism may reach the base of the brain by the lymphatics from the nasal fossæ. It appears to prevail most frequently among

soldiers, especially after prolonged marching and, as a rule, during the winter and spring months. It attacks all ages indiscriminately, but most frequently children and the young from fifteen to thirty years. Cerebro-spinal meningitis is generally fatal, the average number of deaths being about sixty per cent.

Symptoms.—In most cases the patient is suddenly attacked by headache, nausea, violent vomiting, and acute pain in the neck and down the spine. The tongue may be either clean or furred, or, as the disease progresses, dry. Chills sometimes occur, as also vertigo and diarrhoea; but constipation is more frequently present. The urine is albuminous and may contain blood. The slightest noise or movement of the body greatly increases the pain. Tetanic spasms not infrequently occur. There is rigidity of the muscles of the back and neck. The head is drawn back so that the occiput may be between the shoulders. Orthotonos, and more rarely opisthotonos, may be present. The patient frequently holds his head back to relieve the pain in the neck, which at the same time is quite rigid. The temperature is seldom high, 100° to 103° ; the pulse, natural or slightly quickened in adults, is liable to considerable fluctuations in children. A herpetic eruption frequently breaks out round the mouth, but may also appear on the trunk or extremities. Urticaria may also be present and in some cases the eruption is purpuric or petechial. The eruption distinguishes this disease from other forms of meningitis. All the symptoms—delirium, sleeplessness, &c.—which mark the presence of inflammation of the membranes of the brain and of the spinal cord are present. Strabismus is a frequent and important symptom. Inflammation of the eyeball, ending in suppuration and entire destruction of the organ, sometimes occurs. The course is very variable, sometimes causing death in a few hours and sometimes lasting for months.

Diagnosis.—Do not mistake for tubercular meningitis, pneumonia and other acute diseases—typhus, typhoid fever, &c. The diagnosis in all cases is not easy, but a review of all the symptoms will help to avoid mistakes. Lumbar puncture should always be made, and a careful bacteriological examination will do much to clear up the diagnosis.

Treatment.—So uncertain in their curative effects are the various methods of treatment that have been suggested for cerebro-spinal meningitis that the practitioner will find it better to rely on general principles and adopt those measures which the case appears to demand—thus, ice to the head, chloral, morphia, or bromide of potassium to relieve pain or uneasiness, &c. Purgatives to relieve the bowels, if necessary, and mustard sinapism to the back of the neck, or the application of belladonna to the same part, may be tried. Opium and quinin in large doses, frequently repeated, have found favour in America. Stillé advocates the use of 1 gr. of opium every hour in severe cases, and every two hours in cases of moderate severity. Ziemssen recommends the hypodermic injection of morphin in doses of $\frac{1}{3}$ to $\frac{1}{2}$ gr. in adults, and a combination of bromide and morphia is useful for controlling the spasms. The diet must be nutritious, consisting of milk, eggs switched in milk, strong soups, and beef-tea, and in cases where it is found difficult to feed the patient the stomach tube may be employed. Any sign of cardiac failure is best treated by the free administration of whisky or brandy. Iodide of potash is the only remedy of accredited value for treatment in the later stages of the disease in favourable cases, with the hope that it may aid in the removal of thickenings of the meninges or inflammatory deposits.

EPIDEMIC CHOLERA.

Lat. *Cholera pestifera*. Fr. *Choléra Asiatique*. Ger. *Cholera* ;
Asiatische Cholera.

DEFINITION.—An epidemic disease, characterised by vomiting and purging, with evacuations like rice-water, accompanied with cramps, and resulting in rapid collapse.

Etiology.—Cholera has been known in India from a remote period, whence it gradually spread to China and later to Europe, visiting England in 1831. Since then there have been several outbreaks in Europe and America. An immense amount of information has been gathered together bearing upon its different aspects, but prior to 1883 little or nothing was known as to the real nature of its virus. Koch was sent to Egypt in that year to investigate the disease, and in the following year announced his discovery of the comma bacillus, or cholera spirillum, which he regarded as the specific germ. Corroboration of his views soon came from other quarters, although it was not until the disease revisited Europe in 1892, 1893 and 1895 that the opportunity was given for its extensive investigation. Many of the workers agreed with Koch that the cholera organism was a specific spirillum which showed a considerable variability of form, but others held that the variations were too great to admit of this view, and that they indicated rather distinct species or different vibrios. The former view (Koch's), that cholera is a strictly specific disease always caused by one and the same organism, has gradually gained ground, and is now fairly generally believed in. They are small organisms, distinctly curved, occurring singly or in pairs, with the curves in opposite directions so as to give an S-shape. They are actively motile and are present in the intestines and in the stools, particularly in the

characteristic rice-water evacuations, in enormous numbers—a valuable diagnostic point. As in the case of typhoid, the organisms leave the body only in the intestinal and urinary discharges, and their chief method of entrance into the body is, like typhoid, through contamination of the drinking water. They may also enter with the food. Milk, for instance, may be contaminated by flies, and vegetables by being washed in impure water. They remain alive in pure or in sewage water for a very considerable time, though they do not multiply to any extent. They flourish abundantly on moist linen. Generally speaking, they require for growth a warm temperature, moisture, a plentiful supply of oxygen and a large proportion of organic matter. These, and whatever other favourable conditions outside the body are necessary for the growth of the organism, are rarely found combined, since the endemic disease has a very limited geographical distribution. The spirilla are readily killed by drying and hence their distribution and spread by the air is not likely to take place. The disease spreads along the lines of human intercourse and traffic. Individual susceptibility has, as in other infectious diseases, considerable influence, and so has fever and other depressing mental emotions. A porous soil is favourable to the spread of the disease, while an impervious one is not, probably because the falling ground-water leaves conditions favourable to the growth of the spirillum, viz., moisture, air and organic matter, and facilitates its access to wells.

Morbid Anatomy.—There are no strikingly characteristic changes. The temperature often rises considerably after death, and the early occurring rigor mortis may cause muscular movements and distortions. The serous coat of the small intestines may be slightly injected, and the mucous coat more markedly so, or even necrosed in the more chronic cases. The lumen contains some turbid

watery fluid, similar to the rice-water material passed during life. These changes are most marked towards the lower ileum. In the rare cases, known as cholera sicca, in which there is no discharge during life, the intestine is distended with similar fluid contents. There is cloudy swelling of the liver and kidney, and the epithelium of the convoluted tubules may be greatly degenerated or even necrosed. The bacilli are found only in the intestine, being present abundantly, almost in pure culture, in the intestinal contents and walls, but not extending deeply, being limited to the epithelial lining and Lieberkuehn's follicles. They lie in great numbers between the columnar epithelium and the basement membrane, and cause the separation of the former. The epithelium is often found after death to be extensively denuded, but probably this does not take place much during life. The spirilla, unlike those of typhoid, multiply rapidly in the intestine, and produce rapidly-acting toxins, which account for the general disturbances. The blood is thick, tarry and dark.

Symptoms.—The period of incubation appears to vary from twelve hours to two or three days.

PREMONITORY STAGE.—In *some* cases an attack of cholera is preceded by simple diarrhoea and vomiting. The latter generally begins later than the former. After this condition has continued for a period more or less prolonged, the specific symptoms of the disease make their appearance. Of the cases of cholera treated in St Bartholomew's during the epidemic of 1866, premonitory symptoms were absent in 43·1 per cent. of fatal cases, and in 21·6 per cent. of non-fatal cases. "My experience," says Dr Church, "would certainly lead me to say that premonitory symptoms are less frequently present in the severer than in the milder cases." In *other* cases, however, the attack comes on suddenly, and rapidly passes into the algide form, death occurring,

according to Dr Gavin Milroy, "within little more than five minutes" in hale and hearty men. In cases not so rapidly fatal as those just mentioned, the premonitory symptoms, as above-mentioned, are simple diarrhœa, accompanied with vomiting, and may last from two to twelve hours or longer. As the disease progresses, the bowel discharges suddenly become copious, consisting of a thin liquid containing a large quantity of white flocculent particles, which give to the discharges an appearance not unlike whey or rice-water; hence the name "rice-water stools" of cholera. The vomiting and purging now become incessant; the act of evacuation is not always accompanied with pain, but often with a sense of relief. The absence of *bile* in the vomited matter and in the evacuations, together with the marked tendency to early collapse, distinguish this disease from English or summer cholera, prevalent towards the end of the summer months and the beginning of autumn. The urine is diminished in quantity, is albuminous, or sometimes entirely suppressed, the bladder being found empty on examination after death. Painful contractions—*cramps*—of the muscles of the soles of the feet, calves and abdominal walls are always more or less present. There is great thirst and restlessness.

STAGE OF COLLAPSE.—If the disease be not early checked, it passes into the *algide* or *cyanosed stage*, ending in fatal collapse. The temperature of the body rapidly sinks, the breathing is increased in frequency and the pulse accelerated and feeble, sometimes almost imperceptible. The skin assumes a bluish-slaty colour, and it becomes clammy or covered with sweat, and even the breath feels cold to the hand. A fall in the temperature of the body below 94·5° F. generally indicates speedy death. The face assumes a peculiar pinched expression, the eyes are sunken, the corneæ are flattened, the complexion muddy-looking, and the whole expression of the face is known as the *facies cholericæ*. The secretion of milk in suckling women is

not decreased; on the contrary it may be even increased. The catamenia are also not arrested. These and the sweat are the only secretions which are not arrested. Liquid motions may still be passed occasionally, and cramps often continue. There is great and profound debility. Collapse often ends in death, and it is in this stage that death most frequently occurs. The duration of an attack of cholera is usually from three to eighteen hours from the commencement of the symptoms.

STAGE OF REACTION.—Should the disease, however, be arrested, the *stage of reaction*, associated with more or less febrile movement, sets in, the purging gradually diminishes, the breathing becomes slower, and the pulse becomes again perceptible at the wrist and gets stronger. The blueness, coldness, and clamminess and shrinking give place to fullness and warmth. A rash not unlike roseola or urticaria may occur during this stage over the backs of the hands and forearms, and may even extend to the trunk. Even when this stage has most favourably set in the patient is not free from danger, as sudden collapses are by no means uncommon. During reaction Goodeve says that the urine, which may be delayed for many hours, is high-coloured, acid, and possesses a strong animal odour at first, and later becomes copious and watery.

The mental torpor disappears and complete recovery may take place within a few days.

Diagnosis, &c.—This must be obtained from a study of the symptoms just given, and errors of diagnosis can only occur in sporadic cases. In India, poisoning must not be mistaken for cholera, and this caution is not altogether unnecessary in England. Several fatal cases described as sporadic attacks of cholera, *cholera nostras* or *cholera Europea*, have subsequently been discovered to be due to arsenical poisoning to obtain the insurance on the lives of

the persons poisoned, and which the insurance offices, on the good faith of the medical practitioner, have paid. Ptomaine poisoning and mushroom poisoning may also resemble cholera. The presence of the comma bacillus in the stools is pathognomonic.

The **Prognosis** should be very guarded. Fifty per cent. is an average death rate, but it is often exceeded, especially in the early stage of an outbreak.

Treatment.—This is both prophylactic and therapeutic. Every case of incipient diarrhoea during an epidemic of cholera should, if possible, be arrested at once, and “house-to-house visitation,” conducted by properly instructed persons, must be organised. Cholera hospitals should be erected, and the discharges from patients in them should in no case be allowed to enter the common sewers, but should be burnt or disinfected. All infected clothes must be disinfected before being washed. Carbolic acid and chloride of lime should be used freely as disinfectants. The cisterns and drains should be examined and disinfectants used in the latter. Quarantine has in many cases failed to prevent its introduction into a country, mainly from its establishment engendering carelessness as regards sanitary precautions.

Perhaps one of the most important things is to draw up simple rules for the use of those exposed to the risk of an epidemic of cholera. The boiling of milk and water should be insisted on, and all vegetables and fruit should be eaten cooked, or if raw must be washed with boiled water. Anything tending to induce attacks of diarrhoea must be most carefully prohibited. Alcoholic excess should be strongly discouraged and the utmost care taken of the health of those exposed to the disease. Haffkine’s immunising experiments with injections of cultures of the cholera bacillus promise good results.

The treatment of the disease itself is very unsatisfactory. Manson holds that there is little use in feeding during

the acute purging and vomiting stage of the disease, but every attempt should be made to check diarrhoea at its earliest commencement and the best drug is opium, while acetate of lead, bismuth in huge doses, and salol are all useful. The appalling thirst may be relieved by iced water or iced effervescing drinks. Irrigation of the bowel has been recommended and also an attempt may be made to wash out the stomach, but the cramps are so severe and the distress so great that the method is often difficult of accomplishment. There is no question that intravenous injections of salt and water are admirable and have often kept the patient in life. About a drachm of common salt and half a drachm of sodium carbonate to each quart of water forms a good mixture; it may be allowed to flow slowly into a vein, about a quart in ten to twenty minutes being given. Improvement of the patient almost invariably ensues, but it may be only temporary and may merely prolong life.

During the collapse stage heat should be applied by hot bottles and the limbs should be rubbed. The cold sweat must be constantly removed by rubbing with warm and dry towels, and as soon as the vomiting ceases, iced champagne, beef-tea, or milk, should be carefully administered.

During the reaction stage feeding must be cautiously increased, the kidneys gently stimulated by fomentations over the loins, and any undue rise of temperature checked by cold sponging. Change of air and general tonic treatment are of special advantage in the process of recovery.

MALTA FEVER.

Syn. *Mediterranean Fever, Rock Fever, Neapolitan Fever, Danubian Fever, Undulant Fever, &c.*

DEFINITION.—An endemic fever of long duration and irregular course, due to a specific organism and characterised by sweating, rheumatic or neuralgic pains, swelling of the joints, tenderness and enlargement of the spleen, anæmia and liability to relapses.

Etiology.—It is met with in the countries bounding the Mediterranean, in certain parts of India, China, Africa and America. It is caused by the micrococcus melitensis, a small coccus occurring singly and also in short chains here and there. It stains readily with the stronger stains, such as gentian violet or fuchsin. It forms minute, colourless, and transparent droplets on the surface of agar, which become first amber-coloured and then opaque in about four or five days. It grows best at incubation temperatures, but more slowly also at room temperatures, and in all the ordinary media. It may live as a saprophyte in soil polluted by urine, but its path of entrance into the human body is by the ingestion of goat's milk, now proved by Bruce and others to be the method of infection. It is found most abundantly in the spleen.

Pathology.—The spleen is usually much enlarged, friable and pulpy. The liver and kidneys are also congested, and may show foci of hæmorrhage or leucocyte infiltration.

Symptoms.—There is an incubation period of three to ten days, and the onset is either sudden or gradual. There is usually loss of appetite, slight swelling of the cervical
rp.

glands, and headache. Nausea and other symptoms of gastric catarrh come on and persist more or less to the end, but vomiting is not common except in severe cases. He feels ill and feverish. Later, there may be considerable muscular or neuralgic pains, effusion into one or more joints, or acute orchitis. There is always fever, though it is only in severe cases that the temperature reaches 104° or 105°F. and remains continuously high. In the most common type it is undulating, the first attack of fever usually lasting eighteen to twenty-three days, and subsequent ones, of which there may be none, one, or a goodly number, lasting on an average ten days or less. In another type, the intermittent, the temperature is usually normal at night but rises slowly and steadily, to varying heights, without rigor, from midday onwards during the afternoon. The spleen is enlarged and tender. There is a great tendency to bronchial and respiratory catarrhs, and palpitation is common. The pallor of the skin becomes pronounced, there is great wasting, and the hair falls out. Weakness and despondency, especially after several relapses, become very great, but when once recovery properly sets in it is usually very rapid.

Prognosis.—It is very favourable, only about two per cent. dying, though it frequently runs a very erratic and prolonged course.

Treatment.—The primary considerations are careful nursing, attention to diet, and treatment of symptoms as they arise. If there be much sweating the patient should sleep in flannels. Joint pains and effusions should be treated by hot fomentations and thereafter wrapping in cotton wool.

Cold sponging is of great value where the temperature exceeds 103° or 104° . Otherwise, general tonics, iron for the anæmia, and a change of climate are generally sufficient to effect a cure.

INFLUENZA.

Lat. *Catarrhus epidemicus*. Fr. *Grippe* Ger. *Grippe*.

DEFINITION.—An epidemic specific fever, marked by some catarrhal symptoms, particularly respiratory and intestinal, and in some epidemics attended with considerable mortality.

In 1729 there “broke out and raged all over Europe, and perhaps the globe, a most universal epidemic catarrh”; and “in 1732–33,” says Dr Guy, quoting from Dr Short, “the most sudden and universally epidemic catarrh that has been in this age, sparing neither rank, sex, or age, old or young, weak or strong, and killing off many hectic and phthisical people.” “In the space of twenty-five years,” says Dr Guy, “four well-marked epidemics of influenza occurred, that is, in the years 1729, 1733, 1737, 1743.” A severe epidemic also occurred in 1847–48, and a notable one in 1889–90, and less severely each year since. The name Russian influenza has been given to the disease, because many of the epidemics have started in Russia.

Etiology.—The specific bacillus is a short, non-motile rod which occurs singly or in clumps. They are generally present in great numbers in the nasal mucus and the sputum, especially in the small greenish-yellow masses, which often contain practically pure cultures. At first they are mostly free, later they are largely within the leucocytes. Their powers of resistance are slight, and the method of infection is chiefly by direct contact by means of mucus, &c. The fatality in some epidemics has been large. Influenza of late years is seldom fatal, however, except in old people, and in those of feeble constitution. Complications are more to be feared than the disease itself. The duration of the attack is from three to six days, unless complicated with bronchitis, pneumonia, &c. The bacilli

may persist in the sputum for weeks after the symptoms have disappeared, especially if any chronic lung mischief be present. They occur abundantly in the pneumonic patches which occur as complications, either alone or with the pneumococcus or with the tubercle bacillus. In the latter case the prognosis is very grave. The influenza bacillus has been found in a few cases of meningitis following as a complication; in others, the pneumococcus is found. The path of infection is probably through the nose. There are no characteristic pathological conditions found after death.

Symptoms.—The symptoms are those of a specific fever of a severe type, but of short duration, with severe catarrh and often bronchitis. Rigors, followed by sudden and severe prostration, with depression of the spirits, are also among the principal signs of this disease. Influenza has been described under several types: thus we have the *nervous* or *neurotic* form, accompanied with intense cephalalgia and some pains in the back and racking pain in the bones. In the rheumatic form of influenza an unusual invasion of the nervous system, central or peripheral, is *prima facie* in favour of an influenza causation and against the probability of an attack of rheumatic fever; the *pulmonary*, accompanied with capillary bronchitis or broncho-pneumonia; the *gastro-intestinal*, beginning with severe colicky pains, vomiting, and diarrhoea; the *nephritic*, accompanied with severe pain in the lumbar region, painful joints, and scanty urine free from albumin. Mania with suicidal tendencies may be developed during the course of an attack of influenza. Dr A. C. Sansom suggests that the *materies morbi* of influenza may produce in the early periods of its activity symptoms closely resembling those of acute rheumatism. The cases in which the signs closely resemble those of acute rheumatism are to be thus differentiated from the latter:—

In treating the disease it is the sequelæ we dread and not so much the fever itself. We guard against these sequelæ or complications by keeping the patients in bed, or at least confined to the house during the attack, and especially keeping them under observation during convalescence for a week or ten days after the fever has subsided.

Antipyrin, antifebrin, quinin, salicylate of soda and phenacetin are the best remedies for relieving the aching pains and reducing the fever. One or other of these cautiously administered, combined with a hot bath, give great comfort to the sufferer, and light but nourishing diet should be ordered at frequent intervals. Cardiac tonics may require to be administered, and, above all, in most cases, diffusible stimulants are of immense service to counteract the great depression which follows the pyretic stage. At this time the dietary should be made as generous as possible, and the patient fed up on nutritious and strengthening soups, jellies and so forth. Strychnin, iron and other tonics are of great value, and in some cases stimulants should be continued until convalescence is thoroughly established. The mild delirium of the fever itself calls for no treatment; while the depression, so often more mental than physical, which follows the fever should be combated by change of air and surroundings and, above all, cheerful society.

DIPHTHERIA.

Lat. *Diphtheria*. Fr. *Diphtherite*. Ger. *Diphtherische*,
Entzündung der Rachenschleimhaut.

DEFINITION—A specific, infectious, and often epidemic disease, with membranous exudation on a mucous surface (generally of the mouth, fauces, and air passages), or occasionally on a wound. It obtains its name from *διφθέρα*, a skin.

This disease appears to be steadily on the increase. To some extent this may be due to greatly improved diagnosis,

form, or in many other ways. Some writers believe that the infection may come from the cow itself, and that a disease of the teats which is infective from cow to cow may convey to the milk the specific bacillus of diphtheria. One of the lower animals, viz., the cat, is said to suffer from true diphtheria, and more recently the horse also, but the disease which has been described under this name in other animals is certainly not a true diphtheria. The somewhat similar disease of the cow, calf, pigeon, &c., is produced by entirely different organisms. Various other organisms are associated with the diphtheria bacillus in human diphtheria—the commonest being the streptococcus. The staphylococcus is also a frequent concomitant, and inasmuch as these organisms appear to go before the diphtheria bacillus it is suggested that they prepare the way for it, and that a preliminary catarrh or similar lesion is necessary before the specific bacillus of diphtheria can catch on. The latter is capable of very great variation in form and virulence, and the name pseudo-diphtheria has been given to it when it has entirely lost its virulence. It is doubtless the case that there is a limit of attenuation which, if once reached, renders the bacillus safe for all time, but it is extremely difficult to determine when this limit is reached. The term pseudo-diphtheria bacillus should not be used to designate this organism, but may be used to distinguish allied organisms, differing in one or other important characters from the Klebs-Loeffler bacillus. One attack of diphtheria does not protect against a second—it seems rather to predispose towards it—nevertheless it is almost certain that a term of immunity of short duration does follow upon an attack. Age is an important predisposing factor, the greatest incidences being between the second and the fifteenth year, and the greatest mortality between the second and the fifth.

Morbid Anatomy.—The tonsils and pillars of the fauces become swollen, reddened, and usually covered

from the pharyngeal or laryngeal form of diphtheria except by bacteriological examination. This occurs especially in scarlet fever. The disease known as membranous croup may be caused by either the diphtheria bacillus, which is the cause in the great majority of cases, or by a streptococcus, and perhaps by other organisms. It is therefore always an infectious disease. There are other changes still to be mentioned which occur in diphtheria. Capillary bronchitis, or broncho-pneumonia, collapse or interstitial emphysema may occur in the lungs. Degenerations occur in the myocardium, explaining the cases of sudden death from heart failure. The kidneys show cloudy swelling or even parenchymatous nephritis.

Symptoms.—The period of incubation varies “from two to seven days, oftenest two.” The invasion of diphtheria is not always well marked. Sometimes the premonitory symptoms are so slight as scarcely to attract attention, and persons have been known to die without the presence of the disease being suspected till a *postmortem* examination revealed the true cause of death. In other cases rigors, followed by headache and some febrile movement, extreme lassitude—an important symptom—a feeling of stiffness about the neck, and difficulty and pain on attempting to swallow are among the early symptoms. Fever is probably an early symptom in all cases. The temperature rarely rises above 103° except in severe cases. The tonsils then become inflamed and swollen. Sometimes deglutition is performed with little pain, even although the disease be very severe and the patient die. Pain on swallowing is therefore no criterion of the extent or severity of the disease. The diphtheritic membrane first appears as one or several small whitish spots on any part of the throat, but most frequently on the tonsil. It varies much as to size, consistence, thickness and adhesion. It usually extends, and rapid extension is a sign of severity. It may

extend in 24 hours over the whole throat and into the larynx and trachea. Its presence in the latter situation is usually first announced by a small dry cough of a peculiar character, resembling croup. The voice becomes affected, the breathing difficult, and each act of respiration produces a kind of whistling sound. The voice, as the disease progresses, may be reduced to a muffled whisper.

The formation of the diphtheritic membrane then commences as above described, and if removed grows again, and even if exfoliated naturally a fresh formation may appear again and again. After it has ceased to grow it is soon cast off, leaving a reddened swollen surface beneath. This usually occurs from the seventh to the tenth day. A relapse may occur and the false membrane reappear at any time before the patient has recovered from the whole disease. Desire for food is entirely lost, patients often evincing the greatest repugnance to take any aliment whatever. The pulse is at times quick, at others moderate, and more or less compressible; when danger threatens, it becomes irregular. Cardiac failure is more apt to occur than during any other virulent disease. It is caused by the action of the diphtheria toxins on the heart. It usually occurs during the height of the disease. The lymphatic glands at the angle of the lower jaw are swollen and may be tender. This rarely occurs before the false membrane is visible, and it is generally proportionate to the severity of the throat affection, but not necessarily to that of the whole disease. Hemorrhage from the nostrils and other mucous membranes, often difficult to arrest, may occur. The variations in temperature are not well marked, and in many cases the temperature is scarcely above the normal. The fever of onset often falls on the second or third day, although the temperature may be a little above normal for a few days longer. In very severe cases the temperature may be even depressed. The urine is scanty, high-coloured, and

generally contains albumen during the early stage of the disease. The amount of albumen varies in different cases, and from day to day in the same case. It is the result of a true nephritis, which generally disappears soon after the disease. During convalescence, paralysis of the muscles which assist in the act of deglutition not infrequently occurs, the paralytic affection sometimes extending to other parts; occasionally the paralysis appears during the disease, even as early as the fourth day. The paralysis that frequently follows an attack of diphtheria begins in the *soft palate and pharynx*; then there may be some affection of the sight, accompanied with squinting and double vision, and followed by paralysis of the lower and sometimes also upper extremities, the muscles regaining their power in the order of their attack. It is usually confined to the fauces, occurring in about one-tenth of all cases of diphtheria. It lasts ten days or a little more, and then gradually disappears. When the limbs are affected recovery is also certain, but it may be delayed for eight months or even longer. The paralysed muscles either retain their bulk or waste, and the electrical reaction may be normal, more probably reduced, and in certain cases the reaction of degeneration may be present and the tendon reflexes frequently disappear. The nerves of sensation and motion are usually simultaneously affected, but the impairment of sensation is more marked than that of motion. Loss of taste or smell and even deafness may occur. The cause of the paralysis is a multiple neuritis brought about by the toxins of the bacillus.

Diagnosis.—It is impossible to be certain about the diagnosis of diphtheria from the appearances presented by the fauces. A bacteriological examination must be made. The busy practitioner has rarely got the time, skill or experience necessary to reliably carry this out, and fortunately he can now obtain the assistance of bacteriological experts in the matter. The sanitary authorities of many

be negative, a fresh swab should at once be taken in suspicious cases, and if it continues to be negative, the case is not one of diphtheria, however much it may resemble it clinically. A negative result from reliable swabs on each of two successive days almost certainly excludes diphtheria.

The Prognosis should always be very guarded, even when the patient appears to be doing very well.

Treatment.—The treatment of diphtheria naturally divides itself into three heads—prophylactic, local, and general.

1. The prophylactic treatment consists in the thorough ventilation of the sickroom and the disinfection of all discharges from the nose and throat, by which means it may be hoped that those in charge of the patient may be safeguarded, while the bestowal of special scrutiny to the condition of house drains and sinks should be routine practice after an outbreak of diphtheria in a dwelling.

2. The local treatment is specially directed to the removal and limitation of the membrane. The throat may be swabbed with the glycerine of carbolic acid (diluted with one or two parts of glycerine), sprayed with a 1 in 2000 solution of perchloride of mercury, a 3 per cent. or 5 per cent. solution of carbolic acid, or gargled with a fairly strong solution of permanganate of potash.

When the nasal mucosa is involved, much benefit will be derived from using the nasal douche. Medicated steam inhalations of carbolic acid or other suitable antiseptics give great relief, and the air of the sickroom may be kept impregnated by using a vaporiser and spirit lamp. Should asphyxia threaten, do not delay the performance of tracheotomy or intubation till the patient is *in extremis*.

3. The general treatment must also be carefully planned and carried out. The subtle influence of the toxin of diphtheria upon the heart muscle, as well as other muscles,

should be kept in view, and absolute rest in bed enjoined at least for one or two weeks, and especially where there is the slightest indication of cardiac failure. The dietary requires equal care, and repeated small quantities of milk, beef-tea, and sustaining soups should be given at frequent intervals, while alcohol is of great value where the heart is causing anxiety. In certain cases vomiting is persistent and alarming and must be treated by rest to the stomach and nutrient enemata. For diphtheritic paralysis strychnin in $\frac{1}{100}$ grain doses and full doses of tincture of belladonna are of much value. Galvanism, Faradism, and massage are useful adjuncts, and electrical stimulation is of special benefit where paralysis of the diaphragm is either threatening or has developed. Death has in some cases been averted by the use of the battery, and artificial respiration may have to be resorted to every two or three hours, or oftener where necessary.

The antitoxin treatment demands special reference, because it has revolutionised our method of treating not merely diphtheria but many other fevers. We use the blood-serum of a horse which has been rendered immune to the toxin of the diphtheria bacillus by repeated injections of either the toxin or a culture of the bacilli themselves. The serum so obtained counteracts the toxin in the patient's blood and also arrests further development of the bacilli in the body. The serum is standardised by units, one normal unit being the quantity sufficient to neutralise 10 lethal doses of the diphtheria toxin administered to a guinea-pig weighing 300 grammes. The amount given hypodermically to a patient varies greatly; from 1000 to 6000 units may be injected in one or several doses with the strictest antiseptic precautions, and this amount may require to be increased, especially if the treatment is not begun at an early date in the disease. Experience seems to show that one large dose, say 6000 units, given early, is better than smaller ones repeated at intervals. The results

have been most satisfactory, the death-rate has been greatly reduced, tracheotomy less frequently called for, and, where required, more uniformly successful in saving life, and the membrane very rarely spreads after the antitoxin has taken effect. Some reaction, erythematous rashes and joint pains may follow the use of the antitoxin, but are rarely troublesome or dangerous.

LARYNGEAL DIPHTHERIA (MEMBRANOUS CROUP).

There has been much discussion as to the true nature of croup, so called on account of the peculiar noise made by the child, but comparatively little progress was made until bacteriology helped us to solve the question. We now believe that croup is not a specific disease at all, but a group of different diseases which may give rise to somewhat similar symptoms. It may be—(1) A catarrhal laryngitis, in which there is no membrane formed, which is not contagious, and is never fatal unless the inflammation spreads to the bronchi. There is alteration in the voice, laryngeal dyspnoea, and laryngeal stridor, with the addition of a peculiar stridulous or laryngeal (croupy) cough, such as is found in all cases of croup. (2) A laryngeal diphtheria, in which there is a false membrane found, which is contagious and nearly always fatal unless the serum treatment be used. These two forms cannot be distinguished during life unless false membranes be discovered by being coughed up or after tracheotomy. In children, it is in the latter way in which the false membrane is usually discovered, since its ejection through the tracheotomy wound is common, while it is rarely coughed up before tracheotomy. A membranous laryngitis may be due to the streptococcus or staphylococcus, in which cases it almost always spreads from the pharynx. It is indistinguishable from diphtheritic laryngitis except by bacteriological examination. (3) A spasmodic laryngitis

in ricketty children, in which recurrent attacks of dyspnoea and stridor take place, generally two or three nights in succession. Laryngeal diphtheria is rarely paroxysmal or spasmodic.

Diagnosis.—Laryngeal diphtheria is caused by the Klebs-Loeffler bacillus, and though the laryngoscope can rarely be used to advantage, yet a swab of the pharyngeal mucus will generally show the specific bacillus and clear up the diagnosis.

Prognosis.—Laryngeal diphtheria is a very grave disease. Before the antitoxic method of treatment was established it was still much more serious, as recovery occurred only in the few rare cases of spontaneous detachment of the membrane and its ejection by coughing, and in a few successful cases of tracheotomy. The antitoxic method of treatment has greatly increased the chances of recovery.

Treatment.—The treatment for this condition is the same as for Diphtheria.

TETANUS.

Syn. Lockjaw.

DEFINITION.—An acute infective disease characterised by tonic spasms of the muscles, particularly those of the face, neck and trunk.

Etiology.—The disease is caused by a specific rod-shaped bacillus with rounded ends. It varies in length, like the bacillus typhosus, but is somewhat slenderer than it, and it possesses similar long wavy flagella, but has far less power of motility, which ceases entirely when spore formation occurs. Unlike the bacillus typhosus it grows only under anaerobic conditions and forms spores. These grow at the end of the bacilli, and being of larger diameter

than the latter, distend them, and give them a characteristic appearance, hence the names drumstick or pin bacillus. It grows both at ordinary and incubation temperatures, and is abundantly present in ordinary garden soils, in the earth around old ruins, in dust, and in manures, particularly in the excrement of horses. Tetanus is liable to follow local injuries, particularly when severe, such as compound fractures which have got contaminated by dirt; but in many cases the injury may be slight, a mere scratch or a puncture, say with a rusty nail. Observation shows that such comparatively slight injuries in the hands and feet are more frequently followed by tetanus than those occurring elsewhere, but this is merely because the former are more likely to be contaminated by dirt. In a few cases, called idiopathic, occurring especially in time of war, no local injury of any kind can be found, but all the same it cannot be excluded, for the poison may enter through an abrasion or injury too slight to attract notice, such as by a disturbed hair follicle. Experiments upon animals have greatly helped to explain these cases, and also why tetanus, though the bacillus is so common, so comparatively rarely follows dirt-contaminated wounds. As already mentioned, the bacillus is an anaerobe, and while it can grow to some extent in the presence of a certain supply of air, it cannot reach its full development except when all oxygen is excluded. The virulence of the poison it produces is greatly diminished when it has grown in the presence of air, and no evil results follow its introduction into animal tissues. The bacillus itself as well as its spores are killed by the healthy tissues, and it is only when, through mechanical bruising or other injury, the bactericidal powers of the tissues are diminished that tetanus follows the entrance of the virulent bacillus or its spores. The introduction of some of the toxin along with the bacillus or its spores acts in the same way, the toxin, like the mechanical bruising, being able to check the bactericidal powers of the tissues

sufficiently to allow of the multiplication and full development of the germs.

Tetanus is most to be feared after wounds where the tissues are partly protected from the air, as in deep and punctured wounds, and it is probable that in many, if not all of them, some aerobic organism accompanies the tetanus bacillus, and by growing in the more superficial parts of the wound prevents entirely any access of air to the tetanus bacillus itself, and thus helps it to reach its full development. Several of the pyogenic cocci, such as the staphylococcus aureus and the streptococcus pyogenes, have been frequently found accompanying the tetanus bacillus in the wounds in cases of tetanus, and it may be that they play an important part in the evolution of the disease. The tetanus bacilli are confined to the tissues of the wounded area; they cannot by themselves penetrate deeper, and are never found during life in the blood or lymph streams, or in the tissues of the body other than those of the wound. Hence the value in practice of thoroughly exposing and draining or even excising the wounded area. The disease is due to the toxin, one of the most virulent of all known toxins, produced by the bacilli. It is slowly produced, but by the time symptoms appear the disease has gained a firm foothold. The symptoms of tetanus, unlike those of diphtheria which are early, appear late, and are due to a special union between the toxin and the nerve cells of the central nervous system having already become established, in virtue of which the toxin is enabled to exert its action. Free toxin circulating in the blood cannot act. It can only do so after it has become united to the nerve cells of certain portions of the brain or spinal cord whilst they still retain their natural relationships, and so strong is this affinity that once these bonds of union have occurred, they cannot be broken by any antitoxin subsequently injected, nor can they become disunited and enter into union with other

nerve cells. If a portion of the brain or spinal cord be rubbed up into an emulsion with a fatal dose of tetanus toxin, and this emulsion injected into an animal, no symptoms of tetanus follow, because the affinity of the toxin for nerve cells is already satisfied. This will not be the case unless the two are brought together before injection, for separate injection of the nerve tissue after the toxin does not prevent the disease. The entrance of the tetanus bacillus, then, into a damaged tissue, from which air is in some way excluded, is necessary for the production of tetanus. In new-born children, tetanus neonatorum, the infection is through the umbilical cord, and in women after parturition through the uterus, for such cases mostly occur in the tropics, either in cases of premature births or postpartum hæmorrhage. The disease as a whole is more prevalent in hot climates and affects males much more frequently than females (about 6 to 1), doubtless because of their greater exposure to injury.

Morbid Anatomy.—There is no constant or characteristic lesion.

Symptoms.—They do not appear for some time after the injury, usually under two weeks, though acute cases show within four or five days or even earlier and very chronic ones may be delayed for several weeks or even months. Stiffness or soreness about the face and neck are generally the first things complained of, though careful observation may reveal an earlier stiffness or slight tonic spasm of the muscles near the seat of infection, suggesting that the poison first travels up the nerve lymphatics to the corresponding nerve cells in the anterior horns, causing an increased excitability. The stiffness is at first slight but generally quickly increases, particularly affecting the muscles of the jaws—a tonic spasm of the masseters—so that the patient has a difficulty in opening the mouth, hence the name lockjaw.

The tonic spasm may extend to or begin in the facial muscles, giving a set expression to the face and increasing the nasio-labial folds, the "risus sardonicus," and also wrinkling the forehead and half closing the eyes. Soon the rigidity increases and extends to the muscles of the trunk, where it may become in severe cases so great as to produce a condition of opisthotonos, in which the muscles of the back are so firmly contracted that the back is arched so that the head and heels alone touch the bed. The abdominal muscles are rigid and board-like, and the muscles of the limbs may also be firmly contracted. Presently paroxysmal contractions usually supervene upon the tonic spasms. It is only exceptionally that tonic spasms or paroxysmal contractions occur alone. As the case progresses the paroxysmal contractions usually increase in frequency and severity and are easily induced by slight external stimuli, such as a sound or draught of air or movement. When the seat of infection is in the head "cephalic tetanus" results, which gives some *paralysis* as well as rigidity of the facial muscles, and occasionally also marked spasm of the pharyngeal muscles. The tonic contractions go on all the time except during sleep, which is, however, often absent. Pain is always present, at first slight but later severe, particularly during the paroxysms, as any one who has experienced cramp can well understand. If there be no local suppurating wound there may be no fever, particularly in moderate cases; but in severe cases with frequent paroxysms the temperature may rise to 105°F. or higher. After death it may be much higher, due, as some think, to the heat generated by the great muscular contraction. The patient generally dies from exhaustion or cardiac failure, or in the acute cases from asphyxia, due to prolonged tonic spasm of the respiratory muscles, the intellect remaining unaffected.

Diagnosis.—From strychnin poisoning, which it most resembles, it is to be distinguished (1) by the history; (2)

by the comparative late appearance of the symptoms, whereas in strychnin poisoning they generally come on within a short period after the poison has been taken; (3) by the longer duration of the case, for in strychnin poisoning the patient is usually dead or recovering within two hours or sooner after the symptoms begin; (4) by the character and distribution of the spasms, for in strychnin poisoning they are violent, convulsive movements, affecting the whole body at once, the jaws being affected late or not at all.

Prognosis.—It is a very fatal disease. The acute cases practically all die within seven days. It is said that the mortality is at least 95 per cent. Death may even occur within 24 hours after the symptoms appear. The chronic cases, on the other hand, often recover. The later the onset of the symptoms after infection, the less severe is the case likely to prove, and there is a fair prospect of recovery (which, however, is always slow) if the patient survives for ten days after the onset of the symptoms. The mortality of idiopathic cases is said to be about 50 per cent.

Treatment.—The treatment of this disease is still in a very unsatisfactory condition, because, as indicated above, acute cases have a very bad prognosis, and it is only where the disease is less acute that recovery becomes probable.

1. Attend to the wound if any definite wound is discoverable. Excision and the most stringent antiseptic treatment give the best results.

2. Secure absolute quietness for the patient in a darkened room, instructing the attendant to avoid shaking the bed or even the floor, and to make no noise of any kind.

3. Feed the patient with the most nourishing food possible. The removal of one or more teeth may permit the stomach tube to be passed, or a narrow tube may be introduced through the nose. Milk, egg-flip, strong

beef-tea and similar nourishment should be administered, and even rectal alimentation may be resorted to when necessary.

4. Restrain or relieve the spasms by sedatives, among the best of which are chloral hydrate, physostigmin, cannabis indica, belladonna, bromide of potash, and hyoscin; while occasionally chloroform narcosis has been used for protracted periods to secure temporary relief and rest.

5. The antitoxin treatment is by no means satisfactory in acute cases, and the experimental results show that when an animal has had three times the minimum lethal dose of the toxin administered, no amount of antitoxin will save life, although it may prolong it. Hence, in an acute case, while not being too sanguine, we should *at once* commence the antitoxin treatment, administering either Tizzone's or Behring's antitoxin. The first dose of Tizzone's antitoxin should be at least 2·25 grammes, and 0·6 grammes for the following doses.

SUB-GROUP (β)—CHRONIC INFECTIVE
DISEASES OF KNOWN BACTERIOLOGY.

TUBERCULOSIS.

DEFINITION—A specific infectious disease characterised by widespread affection of the organs and tissues, or a progressive local destruction of some organ—*e.g.*, the lungs—accompanied by fever and wasting, and usually ending fatally.

Etiology.—The essential cause of tuberculosis is the tubercle bacillus. It is not the only agent at work, but without it all the others are powerless to cause the disease. These subsidiary agents are heredity, age, food, hygiene, occupation, and condition of health, and they all act in one and the same way, viz., by lessening the resisting powers of the body to the attacks of the specific bacillus. The

influence of heredity in this direction is very great. The tendency for certain forms of tuberculosis to run in families is well known, and we generally speak of the members of such families as having inherited a predisposition to the disease. The predisposition lies in the lessened resistance, and different degrees of it are not only inherited but also acquired. Thus poor and insufficient food, ill-ventilated and damp, dark rooms, and various diseases lower the general resisting powers of the body, and render it more prone to attack on the part of an organism so widely spread as the tubercle bacillus. Diabetes mellitus and chronic alcoholism are good instances in point. Predisposition of this kind is chiefly, if not entirely, of an acquired nature. The occupations which may be said to favour tuberculosis are those involving work in close, damp rooms, much exposure, rapid changes of temperature, and breathing a dust-laden air. It is believed that the tubercle bacillus tends to adhere to the dust particles, and hence these occupations are doubly dangerous because they both lessen the resisting powers and bring the attacking bacillus to the weakened tissues. A vitiated air may prove very hurtful, although it does not itself carry the bacillus. It leads to defective nutrition and increased vulnerability on the part of the tissues, and it cannot be too clearly understood that the tubercle bacillus is so widely and plentifully distributed in nature that it is practically impossible, under ordinary conditions of life, to prevent its occasional entrance into the body. Good food and plenty of fresh air and sunlight, on the other hand, lead to a higher condition of nutrition, and hence of resistance against the germ, and its occasional inroads may thus be rendered harmless. Age has an important influence, probably, at first, mainly in the direction of a hereditary predisposition, which may be, however, much modified by environment. We find that, speaking generally, tuberculosis is most common in childhood and early adult life, but that certain of its forms are more prone to occur at certain

ages: thus, bone, joint and lymphatic gland tubercle are commonest in childhood, though met with not infrequently at any age. Pulmonary tubercle is commonest between puberty and twenty-five, and while it is rare before seven and after forty it is not uncommon at other ages. Susceptibility, then, whether hereditary or acquired, or both, and in most cases it is both, plays a most important, though not the all essential part. It cannot be too clearly and emphatically laid down that tuberculosis is not hereditary. The disease, with very rare exceptions, and these only in the youngest infants, is never transmitted from parent to offspring. It is essentially an acquired disease, due to the entrance into the body in postnatal life of the tubercle bacillus; but the predisposition, that is the vulnerability, of the tissues is in part hereditary, and may be so great, in some individuals at least, that under ordinary conditions of life it is impossible at the present day to protect them from invasion by the bacillus, which, finding a suitable nidus, flourishes and multiplies within their tissues and thus sets up the disease. In many cases, however, the hereditary predisposition of the tissues is not in itself so great, but the after conditions of life may gradually increase it until the time comes when the bacillus can settle and flourish within the tissues. It cannot, then, be too emphatically stated that the environment of the individual is of the greatest possible importance in modifying the inherited vulnerability of the tissues, by either increasing or diminishing it, and we are only beginning to realise how great a power for good in this direction lies ready to our hand. The separate tissues of the body, in certain individuals at any rate, are not all equally vulnerable, and hence we find the disease breaking out in certain tissues, and often remaining more or less restricted to them. In one individual it is the bones, in another the lymphatic glands, in another the serous membranes, in another the lungs, and so on, and hence the same form of tuberculosis has a tendency to affect the different members of the same family.

THE TUBERCLE BACILLUS.—This, the essential causative agent, is a short rod of varying length—two to six or more μ . It is of uniform thickness, being thinner than most bacilli, and has distinctly rounded ends. It stains uniformly when young, but irregularly when older, showing clear and stained areas alternately. It is difficult to stain, because of a fatty substance in its capsule, and it retains the stain with considerable tenacity, resisting the ordinary decolorising agents, such as mineral acids (25% H_2SO_4 or 10% HNO_3 or 1% HCL) and alcohol. There are a large number of other bacilli, such as the smegma group, a butter bacillus, the Timothy bacillus of certain fodder grasses, which also resist decolorisation with acid, and are liable to be mistaken for the tubercle bacillus. These “acid fast bacilli,” as they are called, cannot, however, resist decolorisation with alcohol as the tubercle bacillus does. Spores have not yet been indubitably shown to be present in the tubercle bacillus, but certain oval and roundish bodies, demonstrable in some tubercular foci, either along with or without the bacillus, are believed by many to be spore forms of the bacillus. Certain it is that the resisting powers of the bacillus bring it more into line with the spore-bearing than with the non-spore-bearing bacilli.

THE PATH OF ENTRANCE of the Tubercle Bacillus.—It enters the body in three ways, viz., by inoculation, by the air passages, and by the alimentary canal. The first method is rare, and is generally seen on the hands of pathologists. The second is by far the commonest; the bacillus enters with the inspired air, and may lodge in the mouth, the air passages, or reach the lungs, &c. The third method is comparatively rare, but is commoner in children than in adults. The bacillus enters with the food, and may lodge in the mouth or stomach, or, more commonly, in some part of the intestine. The food may be contaminated with the bacillus either of human or animal tuberculosis. Milk is in particular danger of being thus contaminated when the cow

from which it is obtained is the subject of tuberculosis. That human tuberculosis might and did arise in this way was almost universally accepted until Koch made his startling pronouncement at the Tuberculosis Congress in London in 1901, that the bacillus of human tuberculosis did not affect animals, and that the converse, that animal tuberculosis did not affect man, was in all probability also true. Koch's views have so far met with but little acceptance, but the question is at present being most carefully investigated, both in Europe and America.

Morbid Anatomy.—By whatever path the bacilli enter they usually settle in one particular spot, multiply therein and produce their toxins. These toxins diffuse into the tissues around and produce local changes in them, being also, in part at least, absorbed into the circulation, and thus coming to act upon the nerve centres and the system generally. The local action of the toxins upon the connective tissues, in which the bacilli usually settle, is always at first of a constructive kind, similar in nature to that of many other chronic inflammatory irritants. The connective tissue cells of the part are stimulated to growth and reproduction, resulting generally in a cluster of irregular-shaped cells (the "epithelioid" cells), with one or two nuclei. Frequently one of these cells grows to a large size, in which there are many nuclei (the giant cell). Again, the toxins attract leucocytes from the neighbouring blood-vessels, and they join the cluster. Such a cluster of cells is called a tubercle, meaning, originally, a small swelling. The cells therein are often arranged after a definite fashion. The giant cell is towards the centre, and surrounding it are the epithelioid cells, and further out still the leucocytes. These are, in the earliest stage, polymorphonuclear leucocytes and in the subsequent stages lymphocytes. In this way the tubercle first becomes limited in size. It is too small to be seen with the

unaided eye, and it is only when several such tubercles are found close to one another that a visible structure can be seen. When this forms a little body of a greyish colour and semi-translucent appearance, and somewhere about twice the size of an ordinary pin head, it is called a miliary tubercle. It is held by many to be the definite and characteristic lesion of tuberculosis, and in a sense it is, but it must not be understood to imply that in all forms of tuberculosis these definite tubercles are always formed. This will be more easily followed after tracing the second phase or stage of the tubercle. This is a degenerative and destructive one, leading to a more or less extensive area of caseation. It is first seen towards the centre of the tubercle, where the bacilli are most numerous. It then spreads outwards more or less rapidly as the bacilli increase and extend outwards. It is a noticeable feature of all tubercles that blood-vessels never enter them. They are often prominently present at the peripheries of the tubercles, but they never penetrate into their interiors. This lack of nutrition may therefore play a part in bringing about the caseation which sooner or later appears and constitutes such a characteristic feature in most tubercular foci, but it does not play the chief part. This is undoubtedly performed by the toxins of the bacillus. The toxins, therefore, exert a twofold action—an early constructive one and a later destructive one. This difference in action is, in part at any rate, due to their greater concentration as the disease increases. The caseous foci thus produced vary much in size, from tiny areas up to masses the size of walnuts or larger. They are of a whitish-yellow colour, homogeneous appearance, and generally of a softish consistence, not unlike soft cheese. They were formerly known by the name of “yellow tubercle.” Microscopically they show necrosed cells, fat and debris. Both the miliary and the yellow tubercle may occur separately, scattered throughout an organ or tissue in varying numbers, or in irregular

bunch-like clusters; and if the process in either case be sufficiently slow, an ordinary inflammatory reaction, leading to fibrous tissue formation, will occur at their peripheries. In this way the tubercles or collections of tubercles become localised and surrounded by fibrous capsules, often of sufficient thickness to entirely shut off the bacillus and its products from the surrounding tissues. The disease may thus become quiescent or even cured, though it may at some subsequent period be again lit up, the bacilli taking on an active growth and by means of fresh toxins attacking and caseating the circumscribing inflammatory tissues, and thus gaining an entrance into the more vulnerable tissues around. This tendency to local extension is a marked feature of the disease. Any tissue may be thus invaded—*e.g.*, the walls of blood-vessels leading, if thrombosis has not previously occurred, to hæmorrhage; the walls of canals, such as the bronchi, leading to a discharge of the tubercular material in the sputum, and to its extension into neighbouring or distant bronchi by direct conveyance along the walls of the bronchi, or by inhalation. But these methods of extension are not the only ones which prevail. Sooner or later there is usually an invasion of one or other of the circulatory streams. In the great majority of cases it is the lymph-stream, and the tendency of tuberculosis to spread from the area of infection by the neighbouring lymphatics is one of its markedly prominent characteristics. The lymphatic glands thus come frequently to suffer. This is seen most strikingly in the glands of the neck. The bacilli usually reach them from the mouth, gaining an entrance through the tonsils and the pharynx and even the teeth. It is a curious fact that local tuberculosis of the tonsils or of any of the oral structures is a rare condition, and yet tubercular, or “scrofulous,” disease, as it is usually termed, of the glands of the neck is very common, particularly in children. But while oral tuberculosis is rare, ordinary inflammatory oral conditions are very common,

and there is every reason to believe that the tubercle bacillus may lodge upon any such catarrhal surface, and subsequently entering it reach the lymphatics, and thus the glands, without causing any of its specific changes until it reaches these glands. Similarly, tubercular disease of the mesenteric glands, without any sign of a tubercular lesion in the intestine, by which the bacillus in most cases enters, is not infrequently met with. Without attempting to fully explain this curious phenomenon, it may be remarked that the tubercle bacillus has, so far as we know, no power of multiplying until it comes to, and has remained for some time in, a condition of rest. A certain period of incubation is in all probability necessary. The glands of the neck usually become largely converted into caseous masses; extension occurring from gland to gland through direct invasion and destruction of the capsule, or through the lymph-stream. In this way, chains of cervical glands may be affected, and likewise those around the apices of the lung or in the mediastinum, whence direct extension to the lung is an easy step. Invasion of the blood-stream is another and still more serious method of spread. It would happen much more frequently than it does were it not so often prevented by a previous sealing of the vessels by thrombosis. It is generally a small vein which is first entered, and the bacilli are thence carried to the right side of the heart and the lungs. If they pass through the pulmonary capillaries they reach the left heart and the systemic circulation. They may thus be carried to any and every tissue in the body. They may, of course, directly invade the wall of an artery, and thus more immediately reach its area of distribution. They may also reach the blood-stream by the thoracic duct. Large numbers must in one way or another reach the blood, and yet when the blood is examined for them they are to be found therein very rarely indeed, and then only in scanty numbers. They are probably quickly killed in the circulating blood,

and they certainly do not multiply so long as they remain free within the blood. They must become attached to the lining endothelial cells before they can multiply and produce their toxins. This occurs mostly at sharp angles in the vessel walls, such as the place where a branch comes off, and also in the capillaries, where the blood-stream runs slowly. Endothelial cells so attacked, whether in blood-vessels, lymphatics or the lung alveoli, proliferate and desquamate and locally form epithelioid and giant cells. The desquamated endothelial cells and the leucocytes wander out into the perivascular tissues and may carry out the bacilli, which also leave in the fluid exudation. They may also be carried in the blood-stream to distant parts and form fresh tubercular foci.

The invasion of the lungs, or of the system generally, may take place at a very early period after infection. It may indeed be impossible to point to any spot as the primary seat of infection, and an apparently healthy individual may be attacked and cut off within a few days, in which case every, or nearly every, organ in the body is usually studded with miliary tubercles, although there is generally a greater incidence in the lungs, spleen or serous membranes, such as the meninges, than elsewhere. Apart from such cases of rapid general tuberculosis, the disease is at first a localised one. There may be only one primary focus, or many formed practically simultaneously. This is particularly the case in many instances of acute tuberculosis of the lungs, which run a rapid clinical course of a few weeks duration. The lungs are studded with soft caseous masses, varying in size, scattered or grouped in clusters. The appearances and grouping of many of these masses clearly suggest that they are of secondary formation, but there are many others which have all equally the characters of primary, or practically primary, lesions. It is more common to find one, or less frequently two, or a few primary local lesions of old standing, from which there

has been a gradual and slow extension into the tissues around, and after these have been in existence for many months or, more frequently, years, an acute outbreak of acute miliary or caseous tubercle in the same and other organs generally precedes death. No organ or tissue in the body is free from the attacks of the tubercle bacillus, and the appearances presented in each case vary according to the rapidity of the course and the anatomical features of the affected part. They will be referred to later somewhat more in detail in the systematic account of the different systems. A general description only is here given, but it will greatly simplify the detailed descriptions when these are reached. It should be noted that the greater the chronicity of the disease, and it is typically a chronic disease, the greater is the consequent fibrosis, resulting often in much contraction and deformity. A question of great practical and pathological interest in connection with the subsequent softening and breaking down which so often takes place in tubercular lesions, resulting in abscess and cavity formation and purulent discharges, is the part played by the tubercle bacillus in these changes. Thus caseous tubercular glands in the neck, caseous foci in the lungs, joints, bones, &c., so constantly result in the formation of pus that it seems to be a natural stage in the evolution of tuberculosis, and consequently to be due to the continued action of the toxins of the tubercle bacillus itself. On the other hand, may it not be due to other organisms finding their way into a tubercular focus and setting up their specific actions? In support of this view, it is to be noted that it is particularly those tubercular lesions whose anatomical relations make the inroads of the pyogenic organisms' easy which suppurate, and bacteriological examination of the fluids obtained through their artificial or natural puncture often demonstrates the presence of these organisms along with or without the tubercle bacillus. There can be little doubt

that clinically most of the symptoms, perhaps all but those of the early stages, of phthisis and many other tubercular lesions support this view. On the other hand, it is also possible that the tubercle bacillus itself may possess a digestive power, resulting in the formation of fluids practically similar to those more rapidly formed by the ordinary pyogenic organisms, for the contents of uncontaminated cold abscesses, such as the psoas, are generally found to be sterile or to contain the tubercle bacillus alone. It is probable that in many cases of tuberculosis both actions are at work, and that though in their early stages they are examples of pure tubercular infection, in their later stages they are *mixed* infections.

Symptoms.—The general symptoms of tuberculosis are—

(1) Fever, (2) anæmia, and (3) wasting. The other symptoms vary with the organ attacked: thus if the lungs be affected there are generally signs of consolidation or of cavities; if the meninges, there may be head retraction, vomiting and coma, and so on. These will be considered more in detail hereafter.

1. Fever is rarely absent in tuberculosis, particularly when the disease has once obtained a firm footing. It is usually moderate, 101 to 102°F. or so, but its irregularity is its most striking characteristic. It generally shows diurnal rises and remissions, the rises occurring in the afternoon or evening and the remissions in the morning or *vice versa*, and both types may be present at different stages of the disease in the same individual.

2. The anæmia is generally steadily progressive if not combated, and may become so severe as to seriously threaten the life of the patient before the tuberculosis itself actually does so. The blood shows mainly a deficiency of red blood-corpuscles and hæmoglobin; in other words, the characters of an ordinary secondary anæmia, and, generally speaking, its degree is a fair indication of the

severity of the primary mischief—the tuberculosis. There is no leucocytosis in uncomplicated cases of tuberculosis.

3. The wasting is 'not' usually a pronounced feature in the early stages of most cases of tuberculosis, but it is always marked in the later stages of the chronic form. The anæmia and wasting may be due, in part at least, to the other organisms, such as the pyogenic bacteria, which so frequently complicate tuberculosis.

Diagnosis.—Careful attention to the general symptoms, and to the physical signs produced in the implicated organs, will often enable a sure and certain diagnosis to be given. Particularly is this the case when the tubercle bacillus is found in the discharges. Unlike the diphtheria bacillus, for instance, it is an *infallible sign* that the disease, of which it is the cause, is present in the body from which it comes. But in the early stages of tuberculosis and in cases of acute general tuberculosis there is often considerable difficulty, and then a careful examination of the blood for the Widal reaction, for a leucocytosis, &c., will be of the greatest assistance in differentiating it from typhoid fever and other diseases which often resemble it.

Prognosis.—In the past little, if any, hope could be confidently held out to the unfortunate victim of tuberculosis, but in more recent times especially, both pathological and clinical experience have encouraged a much more hopeful view. If early cases be taken in hand in time there is every chance of recovery, and even in the later stages much good may be done, though the chance of ultimate recovery in them is but small. The modern crusade against tuberculosis is much more thorough than anything ever attempted in the past. It attacks the disease from every side, and already the results have been so promising in prolonging life and decreasing the mortality from the disease, that if we continue to make the same rapid strides

in the future, tuberculosis may be almost banished, or at least relegated to the category of rare diseases, within, say, the next fifty years.

Treatment.—Much has been and can still be done in the direction of prophylaxis. While it is true that all tubercular patients are not dangerous to their fellows, cases of active pulmonary tuberculosis should certainly be considered and treated as infectious. The strongest pressure should be brought to bear to deter the subjects of tuberculosis from marrying, and especially is the infected mother a source of great and constant danger to her children, as already indicated in the preceding pages. Sunlight and fresh air are bactericidal, and delicate persons, and especially children, can be shielded from many risks of infection by a wise and yet strong public health department. Everything tending to development of the chest, and, indeed, the well-being of the body as a whole, should be pressed upon the attention of those who are themselves tubercular or who have the care of “delicate” and therefore suitable subjects for the attack of the tubercle bacillus.

How far the correctness of our views of infection through cow's milk and bovine tuberculosis generally is justifiable we are not in a position at present to say; but without treating with any contempt the recent statements made by Koch on the immunity of the human subject from bovine tuberculosis, we ought to enjoin the same strict examination of cows and the rejection of tubercular meat which are at this moment in force. Our present views may be wrong, but we are at least erring on the safe side.

When we are dealing with patients probably tubercular, we should place them under the best possible conditions as regards fresh air, food, exercise and, perhaps most important of all, work; and much may be done to strengthen the patient so as to help him in the battle with the tubercle

bacillus. Dry and pure air, with plenty of sunlight, and residence at a high altitude, or a long sea voyage, have not infrequently turned the fortunes of war in favour of the patient.

We should remember in the course of how many postmortem examinations obsolete tubercular foci are found, even in patients in whom we have never had any reason to suspect the disease, and that as these are a source of danger and might have been lighted up into active disease, the physique not of delicate families merely but of the nation as a whole should be improved by the encouragement of every reasonable form of athletic sport and of healthy outdoor exercise, especially for those whose work necessitates long hours of confinement.

Tubercular disease of the lungs and other organs will call for special description later, and under their respective headings treatment suitable for each will be given. Enough has been said here to indicate the best method of treating tubercular subjects generally. Two points still remain to be mentioned briefly. One is the segregation of tubercular cases who are infectious to their fellows, and the thorough disinfection and destruction of infective sputum expectorated by these patients. Reference to such methods of disinfection will be found incorporated with the treatment of phthisis pulmonalis. The second point to which we must refer is that of the application of tuberculin as a curative agent. It is hardly necessary or possible here to discuss fully the exact action of Koch's tuberculin, or the varieties and derivatives he has more lately introduced as substitutes and improvements upon it. The most recent is Tuberculin R., which is apparently anything but successful. Many other "serums" have been introduced, but none of them are of much value. That an antitoxin, or better still a bactericidal agent, will be discovered to cope with the tubercle bacillus we have every reason to believe, but it is not yet an accomplished fact.

LEPROSY.

Syn. *Elephantiasis Græcorum* ; *Lepra*.

DEFINITION—A very chronic infectious disease due to a specific bacillus, and characterised by granulomatous infiltrations of the skin, nerves, and viscera, leading to trophic changes and generally to death.

Etiology.—While leprosy is met with in every country in the world it is very rare in some—*e.g.*, Britain—and very common in others—*e.g.*, equatorial countries. It is not general in cold and temperate regions, but increases in frequency as the equator is approached. Some districts in Norway are an exception to this statement, for it is more prevalent there than in any other part of Europe. It is caused by a specific bacillus—the bacillus lepræ—which resembles the tubercle bacillus very closely, differing from it, however, in a few minor details, such as, when stained, the greater resistance its colour offers to acids and its tendency to occur in colonies, and within the tissue cells rather than outside them. The bacillus has not yet been cultivated artificially, unless the recent claims of Carrasquilla and Campagna should prove to be well founded. The disease has not been transmitted to any of the lower animals, and is not known to affect any other than man. We are unacquainted with the conditions which predispose to the disease further than that the same is true of it as of other infectious diseases—that all conditions which weaken the health and strength may do so. Heredity has been claimed to play a special part, but this has not yet been proved. It is, however, a suggestive fact that healthy individuals entering a leprous district rarely acquire the disease; on the other hand, the children of lepers are often free from any leprous taint. Transmission from parent to offspring is probably as likely as it is in syphilis. In its acquired form its transmission among adults also closely resembles

that of syphilis, and is thought to be made chiefly through sexual congress. Accidental transmission of either of these two diseases is rare, but Jonathan Hutchinson holds, and has ably advocated the view, that the disease is propagated by eating putrid fish. It may appear at any age; but seldom before the third year of life, and never at birth. The isolation and segregation of lepers has been proved to be the most efficient means of controlling the disease, both in past and present times.

Pathology.—The change in leprosy is widely spread. It consists mainly in the formation of a granulation-like tissue in the subepithelial layers of the skin, mucous membranes and the nerves, and later, as a secondary change, in certain of the organs, such as the liver, spleen and testicles. In consequence of the affection of the nerves their fibres are destroyed, and striking trophic changes follow, leading to atrophy of the skin, muscles and bones. This granulation-like or granulomatous tissue sometimes takes on a mainly nodular or tubercle-like arrangement, and the term "*lepra tuberosa*" or "*tuberculosa*" is used; at other times it is more diffused and leads to more marked sclerotic changes, and the term "*lepra maculo-anæsthetica*" is used. There is, however, in all cases a mixture of the two forms, although the one may greatly predominate over the other. The tubercular form usually begins in the exposed parts of the body, such as the skin of the face, backs of the hands, feet, arms and legs, and later anywhere, as a crop of small nodules, attended by fever, with sometimes a temperature as high as 103° or more and lasting for a day or two. Erythematous patches may accompany or precede the crop of nodules. The nodules may steadily enlarge, and fresh crops may be produced at intervals, or many of the old ones may disappear as new ones appear. They vary in size from a pin head to a walnut or even larger. They show as masses and ridges beneath the skin, often causing much

disfiguration and distortion. In the face the appearance has been spoken of as "leonine"; the ears, nose, mucous membranes of the mouth, pharynx and larynx become thickened. The infiltration affects the subepithelial tissues of the true skin and mucous membranes, and, to a less extent, the deeper tissues. The overlying epithelium may become stretched and fluid may ooze from it, or it may become ulcerated. These ulcers may heal, leaving a scar, or they may extend, causing considerable destruction. Thus the part of the nose supported by cartilage may disappear and the cornea may be infiltrated and ulcerated, and keratitis or iridocyclitis may lead to blindness. The discharges, serous or purulent, from the ulcers may show the bacilli in great numbers. The structure of the granulomatous tissue is characteristic: it is very cellular, the fibrous tissue stroma being very scanty in the earlier stages, but as it grows older there is much more fibrous tissue, until, in the late stages, it shows a dense fibrous and sclerosed tissue. A large number of the cells are of considerable size and of a rounded or oval shape, with a vesicular nucleus and protoplasm which is hyaline and often vacuolated. These are called lepra cells, and their exact nature is doubtful. They have been looked upon both as modified connective tissue cells and as leucocytes, and also as not cells at all but dilated lymphatic spaces. This last is a view which has much to commend it. These lepra cells are densely packed with the bacilli, which are often so crowded together as to obscure their own outlines and the structure of the cells. The bacilli are comparatively scanty elsewhere—in the lymphatic spaces around, in the spindle-shaped connective tissue cells, in the endothelial cells, and in the walls of the blood-vessels. The vascularity of the leprous nodule is a striking feature when compared with the non-vascular tubercle nodule, and caseation, so typical of the latter, never occurs. The bacilli spread from the nodules by the lymphatics to the nearest lymphatic

glands, which, though always enlarged, never suppurate, and are found to contain the bacilli in enormous numbers. They spread also by the blood-vessels, and they have been found in the blood, both during life and after death, chiefly within the leucocytes, but also in the plasma. They may thus be widely distributed throughout the body and may be found within any of the internal organs—except the brain and spinal cord, kidneys, bones, muscles, and walls of alimentary canal—always, however, in scanty numbers, and generally in the walls of the capillaries.

The maculo-anæsthetic form begins as an erythematous or pigmented patch in the skin, often of considerable size. The onset may be gradual or sudden, and is accompanied by fever. Later the central parts of the patches become pale and anæsthetic, while the peripheral parts, which may be a little elevated, become livid. The eruption is often, but not by any means always, symmetrical. The nerves in the regions affected are diffusely infiltrated with similar granulo-matous tissue to that seen in the tubercular variety, and may even show visible and palpable nodular thickenings. Usually the process is much slower than in the tubercular form, and gradually leads to much sclerosis, with destruction of the nerve fibres. This leads to neuralgias, loss of sensation, and loss of trophic influence, and hence the parts affected become atrophied and distorted, often to a remarkable degree. Exfoliation, necrosis, and separation of parts are apt to occur. The bacilli can often be detected with difficulty in the sclerosed nerves, and their numbers are generally in direct relation to the rapidity of the process, hence they are very scanty in the typically chronic cases. Their method of spread by the lymphatics to the neighbouring glands and by the blood is the same as in the last form. It is to be carefully remembered that most cases of leprosy show a combination of these two forms, for the nodular variety is always accompanied by changes in the nerves, although in the anæsthetic variety the evidence of

distinct nodules is often slight. The disease may steadily progress or remain quiescent for long periods. Waxy disease often supervenes where the ulceration is great, and true tuberculosis often ensues and carries off the sufferer. The view has indeed been advanced that leprosy is but a modified form of tuberculosis but it is not supported by evidence.

Symptoms.—These are practically a recapitulation of the evolution of the pathological lesions above described. There may be no premonitory symptoms; or a period of general ill health, lasting for months, or even years, may be present before any distinct leprosy lesion appears. Even after leprosy shows itself and the lesions have become widely distributed, the patient often enjoys fairly good general health, it may be for years. This shows that the general toxic absorption must be of the slightest, though the local irritative effects are so marked. Lepers frequently carry on their duties of life for years after the beginning of the disease, and their muscular power though weakened is not otherwise injured. Loss of sensation often causes them to injure themselves with hot water, &c., and favours the formation of ulcers, sometimes of a very destructive nature, upon their hands, feet, &c. Sight, smell and taste may be lost, the hands and feet may be mere stumps, and altogether in his last stages the leper often presents a sickening and pitiable spectacle.

Diagnosis.—There is generally very little difficulty, and all doubt may be easily cleared up by finding the bacilli in the suspicious nodule or patch.

Prognosis.—Recovery may take place through ulceration and cicatrization, but great caution has to be exercised, since the disease often breaks out again after long intervals. It is a very chronic disease, rarely ever in the macular form,

which is the more rapid, lasting less than eight to ten years; while, when the other form predominates, it may last twice as long or much longer, even forty years.

Treatment.—Much benefit is obtained in the direction of the greater comfort of those afflicted with either of the forms of this disease by strict attention to hygiene, exercise, frequent bathing, good food, especially fresh meat and vegetables, warm clothing and the use of general tonics, &c., especially cod-liver oil.

There is no certain specific. Chaulmoogra oil taken in capsules (5-40 drops in increasing doses) is much lauded in some leper settlements, and is believed to afford relief to the pains and improve the general health. Other oils (gurjun, chasjew, &c.), used both internally and externally, do not appear to be so satisfactory. Salicylates and salol have been tried with doubtful benefit. Recently Calmette's anti-venene in 20-30 c.c., administered subcutaneously, appears to have had a beneficial effect in a few cases, but the results are not conclusive. Segregation should be insisted on, and especially in cases where there is much ulceration the lepers ought to be considered "infectious."

ACTINOMYCOSIS.

DEFINITION—Actinomycosis, from *ἀκτίς* a ray, and *μυκήσ* a mushroom or fungus, is an infectious disease, due to the actinomyces or ray fungus, especially affecting cattle but may also be found in man.

Actinomycosis is a wide-spread disease among cattle. It also affects swine, sheep and horses, and is not uncommon in man. It is inoculable into many other animals, such as rabbits, guinea-pigs, dogs, &c.

Etiology.—The exciting cause is a pleomorphous organism, the actinomyces, formerly considered to be a fungus, and known as the ray fungus, but now generally held to belong to the higher streptothrix division of the bacteria. It is generally met with in the form of rods of varying length, which grow out into long, thin, wavy, branching filaments, interlacing more or less densely with one another to form a little mass or colony, but showing generally a radiating arrangement towards the margin. Coccal forms are mixed in varying numbers with the filaments, and club-shaped forms are often seen at the ends of the filaments especially in the older colonies. The cocci are probably the young forms of the organism, many authorities regarding them as spores, and the clubs are hyaline swellings of the terminal part of the sheaths of the filaments. Its cultivation is easily obtained on several media, though it grows slowly on them all. The organism is found in great numbers in the centre of the tumor-like masses or abscesses to which it gives rise. Its method of entrance into the body is not definitely established, but there is strong evidence to show that it most frequently does so with grains of certain cereals, particularly barley. It has been found growing around fragments of such grains embedded in the tissues in the ox, pig, and even in man, not only in the mouth—*e.g.*, the tonsil, the hollows of decayed teeth—but also in intestinal lesions; but it has never been found outside the body and cultivated. In man contagium must enter by the mouth in most cases, as in over half of the cases the primary lesion is in the head and neck or tongue, and in fully a quarter more of the cases in some part of the gastro-intestinal tract or its surrounding tissues. In other cases it may be inhaled, as the primary lesion is in relation to the respiratory tract. In a small number it is in the skin, probably by direct inoculation. It has been known to enter by the reproductive system, and in a few cases its method of entrance is obscure.

Morbid Anatomy.—The organism gives rise to an inflammatory nodule of the granulomatous type, showing round cells, epithelioid cells, and a few small giant cells. Its rate of growth varies, but it is always more or less chronic and generally very prolonged. The granulomatous nodules coalesce to form large inflammatory swellings. This is strikingly seen in cattle, as exhibited by the greatly enlarged and tumor-like swellings which it produces in the jaws and tongue ("woody tongue"). In man this formative aspect is not generally so prominent, though it is usually well marked in the lesions of the head and neck and subcutaneous tissues generally, where it takes the form of large nodular and irregular indurated swellings, strongly resembling malignant growths. Sooner or later portions of the growth soften and break down, forming pus which reaches the surface and is discharged, leaving sinuses, often of great length, which continue to discharge a purulent fluid and whose margins are often surrounded by pouting granulations. Suppuration comes on much earlier when an internal organ, such as the liver or lung, particularly the latter, is affected. In the liver an irregular infiltrating mass is formed which on section shows the characteristic appearance of a spongy or honeycomb-like structure, whose spaces are filled with greenish-yellowish white slimy pus separated by more or less dense fibrous walls. In the lungs it may form a similar though smaller tumor-like mass, in which cavity formation is even earlier. The cavities are filled at first with thick puriform material, which may be discharged through the bronchi, leaving discharging cavities with dense fibrous walls, not unlike those of phthisis. In all cases the growth tends to spread by continuity, and this is more marked the more chronic the course. Thus it spreads from the œsophagus to the mediastinum and lungs, from the lungs to the pleura, from the intestine, where it forms collections of abscesses and fibrous tissues in the submucous and other coats, to the peritoneum and sub-peritoneal

cellular tissues, and may form large purulent indurated masses in the region of the cæcum (probably spreading from the vermiform appendix) or behind the rectum. It may reach the liver in this way and in course of time involve the diaphragm and base of lung. There is no tendency to spread by the lymphatics, but a blood distribution is often seen, particularly in the lungs, which may be studded with minute nodules, like miliary tubercle, or show metastatic purulent deposits. Occasionally a bronchitis and peri-bronchitis alone has been seen. In the skin soft tumor-like growths are found, which are apt to be mistaken for tubercle or sarcoma until the skin breaks and the pus shows their true nature. The pus in all cases contains, generally in great numbers, the actinomyces colonies. It should be mixed with saline solution and spread out on a slide and held up to the light, when the colonies will be seen as small masses of varying size, from a pin head downwards, and variously coloured, according to their age, the youngest being of a semi-translucent grey or greenish-grey tint and the older ones yellow, often saffron-yellow or greenish, or even black. Microscopic examination of fresh and stained colonies will show the characteristic clubs and filaments.

Symptoms.—In visible actinomycosis there is the presence of the tumor mass with its above-mentioned characters. Various pressure results may be given, according to its site. In pulmonary actinomycosis there may be cough, especially in the bronchitic type, and the sputum may show the colonies. In the intestinal and other internal forms there are no characteristic symptoms. There is generally in all cases moderate fever and wasting, such as is present in any long-standing suppuration.

Diagnosis.—A certain diagnosis cannot be made until the characteristic colonies are found in the pus or breaking

down granulation tissue. The tumor masses so closely resemble sarcoma and epithelioma, that the distinction cannot otherwise be made. In visceral actinomycosis the diagnosis is still more difficult, and cannot be made unless the colonies be found in the sputum or fæces, or in the purulent discharge after the formation of a sinus.

Prognosis.—It was formerly and still is a very serious malady, but many cases recover under appropriate surgical or medicinal treatment.

Treatment.—When the disease can be attacked with the knife it should be used, or the diseased parts removed by means of Volkman's spoon. Paquelin's cautery may also be tried, with the subsequent application of antiseptic dressings. For the internal manifestations of the disease little can be done; but it appears from recent reports that the iodide of potassium in doses of from 30 to 60 grains a day is of actual benefit in the treatment of this disease. The undoubted success of this drug in animals suffering from actinomycosis induced an experimental administration in man, and with fortunate results in a certain number of recorded cases.

MYCETOMA OR MADURA FOOT.

This is a mycotic affection of the subcutaneous tissues of the foot (occasionally the hand), met with chiefly in India, very similar to actinomycosis. The foot is greatly swollen and brawnily indurated, with many sinuses showing pouting granulations. It is caused by a mycelium which closely resembles the actinomyces, of which many authorities regard it as a variety. It forms similar colonies, either colourless or black, which are present in the walls of the sinus, the pouting granulations, and the discharges.

SUB-GROUP (γ)—*SEPTIC DISEASES OF KNOWN BACTERIOLOGY.*

SAPRÆMIA, SEPTICÆMIA, AND PYÆMIA.

These three terms are now in common use to express different forms of blood poisoning. Septicæmia was formerly made to include Sapræmia, but this use of the term is not correct.

SAPRÆMIA.

Sapræmia indicates the presence in the system of the poisonous products of bacteria. They may enter from without, having been formed outside the body, or they may enter from a local wound or cavity of the body, where they are manufactured. Diphtheria and tetanus are good examples. In these two diseases the bacilli remain within the tissues of the local lesion, and do not, unless under exceptional circumstances, penetrate more deeply or invade the neighbouring tissues or organs or the circulation. Their toxins, on the other hand, are freely absorbed into the tissues and fluids of the system, and give rise to characteristic general effects. The severity of these effects will depend entirely upon the amount of the respective poison which is absorbed by the system, exactly as after the administration of strychnin or any other well-known poison. The poison does not multiply after its absorption, and once it has expended its force, the system, if not overcome, will begin to recover unless it continues to receive fresh doses from the local area of manufacture. Clear out this local area by destroying all the bacteria therein and washing away any toxins not yet absorbed and the system has merely to cope with the dose of poison previously absorbed. The bacteria themselves, apart from their being the producers of the toxins, have no influence upon the condition. They

may, indeed, be unable to live within the living body, being purely saprophytic, and this is the case in many of them. The effects produced by the different toxins upon the body and its tissues naturally vary greatly. They may be very acute and severe, as in the case of tetanus, or may be so very slight as to be in themselves scarcely recognisable, and are chiefly demonstrable in the lowered vitality of the tissues, which now readily become a prey to attacks of true, or, to use a better term, more powerful, pathogenic organisms, which formerly they were able to withstand. This influence of toxins upon the growth of bacteria is a most important one. It is seen not only in a number of saprophytic organisms preparing the way for pathogenic ones, but also in one pathogenic microbe preparing the way for another, or even for itself; for few influences are more powerful than the toxins of tetanus or diphtheria in preparing the ground for the growth and development of their respective micro-organisms.

In practice the condition of sapræmia is not at all infrequently seen, as in localised abscesses, septic wounds, or in cavities with decomposition of their contents, where the micro-organisms present remain restricted to the local lesion, while free absorption into the system of their poisonous products takes place.

Symptoms.—These vary greatly according to the nature of the toxins produced in each case, but they possess certain features in common. They tend to upset the system, giving rise to headache, thirst, vomiting, gastro-intestinal irritation, fever, a quickened pulse and respiration, muscular weakness, and collapse.

Prognosis.—In ordinary cases, where the source of the trouble can be got at and removed, the outlook is favourable and recovery is to be expected, but even here it will depend somewhat upon how far the tissues have been

already damaged, as they may now be more prone to fresh microbic attacks.

SEPTICÆMIA.

This indicates a condition generally more serious than sapræmia, in so far as the system shows not only the toxin absorption, but also the invasion of active living germs into the vascular system. The germs pass from their point of entrance into the circulation, and are carried to every part of the system. They multiply therein, and at every point produce their toxins, the manufacture of which is thus no longer local, but general throughout the system. There is no limitation of the dose which the system may absorb, and the wide-spread and general distribution of the germs makes it impossible to reach and exterminate them. The germs are present in the circulating blood, and unless this can be demonstrated the condition of septicæmia cannot be established. It is surprising how very rarely germs are found in the blood of the peripheral circulation in cases which present all the appearances of septicæmia, the reason for this being, that no matter how plentifully they invade the blood, they soon leave the peripheral circulation and tend to accumulate in the capillaries of the internal organs and to pass through the endothelium into the surrounding structures. Thus in cases in which examination of the peripheral blood during life gives few or no organisms, the capillaries of the kidney or other organs may show them in great numbers in the lumen and in their endothelial lining.

Failure to demonstrate the germs in the blood of the peripheral circulation has been more frequent than it ought, due doubtless to imperfect technique. More recent investigations, where a large quantity of blood (2 to 5 ccm.) has been withdrawn and added at once to culture tubes of broth, have given a far greater percentage of positive results.

Anthrax in animals and pneumococcal infection in guinea-pigs are excellent instances of septicæmia, as in both these diseases the organisms are present in great numbers in the blood; and in the human subject suppurative conditions are the most common examples.

Symptoms.—They must obviously present a close resemblance to those of sapræmia, as in each case they entirely depend upon the toxins. They differ markedly, however, in being but little affected by the clearing out and proper treatment of any local wound. They are often, also, slower of commencing, as the germs cannot produce their toxins until they have succeeded in affecting a lodgment in the tissues.

Prognosis.—This will depend entirely upon the gravity of the symptoms. It is bad in all severe cases, though slighter forms, especially in healthy individuals, often recover.

PYÆMIA.

The formation of one or more small or large foci of suppuration, arising in some way from the primary lesion, is the distinguishing feature of pyæmia. These secondary suppurations and abscesses arise in several ways, of which the best known is where a septic phlebitis occurs in a vein in the neighbourhood of a primary lesion, as in a septic wound, and portions of the consequent septic thrombus become softened and detached, forming septic emboli, which are carried by the venous circulation to the right side of the heart and hence to the lungs. They may stick in some branch of the pulmonary artery or capillaries and cause abscess formation in the lungs, or, if small enough, they may pass through the lung capillaries, and after being carried to the left side of the heart pass into the systemic circulation and thus reach the spleen, kidney, brain, &c., and give rise

to abscesses in one or other of these organs. In ulcerative endocarditis, detached bits of the vegetations act in the same way. There are at least other two ways in which the blood may convey the germs which give rise to the secondary suppurations characteristic of pyæmia. The first of these is where the pyogenic germs pass directly from the local lesion into the blood without the intervention of a thrombosis, and are carried by the circulation to some area of lowered vitality or specially suitable nidus, in which they multiply and develop, and thus produce an abscess. The second is where they spread along the vessel wall, not by means of the circulating blood so much as by actual continuity in the vessel wall. This is well exemplified in the portal vein when a lesion of the gastro-intestinal tract causes a septic phlebitis and thrombosis of some branch of the portal vein, and then the suppurative process extends in the walls and lumen to the main trunk itself, and to its branches within the liver, causing the disease known as suppurative pyelophlebitis. Pyæmia may, however, be set up in other ways than by the blood, viz., by the lymphatics and by natural channels, such as the ureters and bile duct. In the former the microbes pass along the lymphatic trunks, which may or may not become inflamed (lymphangitis), and settle in the lymphatic glands, causing suppuration therein. It is especially in diffuse cellulitis that this form of pyæmia is seen, and the causative organism is most frequently the streptococcus pyogenes. In the latter the microbes pass directly from the primary lesion, lower down, say, in the bowel or bladder, along the bile duct or ureter, and settling in the liver or kidney, produce abscesses.

Symptoms.—These will vary with the nature of the primary wound and the size and numbers of the metastatic abscesses and the amount of toxic absorption which follows from them. In cases with small, rapidly-formed and multiple abscesses the symptoms will naturally closely resemble those

of septicæmia; while in cases with large and few abscesses of slow formation they will be less severe, slower in coming on, and most marked during the period of the abscess formation. They generally appear within the first week of injury or operation. Rigors and rises of temperature to 103°F . or higher may occur during the formation of an abscess, followed by profuse sweating, after which the temperature may fall to normal, or even below it. There are the usual febrile symptoms, and skin eruptions of a rosy-red, papular or pustular character sometimes occur. The metastatic abscesses cannot usually be localised unless they are of some size, when pain and swelling may direct attention to their place of formation. In the more chronic cases they are to be looked for especially in and about the joints and serous cavities of the body.

Prognosis.—This is always grave, as although the patient's condition may be favourable for a time, it may become serious at any moment, through the incidence of some grave complication, such as peritonitis, meningitis, pericarditis, &c. If the primary lesion can be got at and cleared out, and the metastatic abscess take a favourable course and no vital organ be affected, then the patient may recover; but otherwise he usually passes into the typhoid state, or dies of exhaustion, or becomes more actively delirious and succumbs to rapid toxæmia or meningitis, &c. Death usually occurs in from eight to twelve days.

Treatment.—The principles of medical treatment of these conditions are—(1) To support the patient's strength and so help him to withstand the toxins or organisms present in his blood; (2) to remove the cause where this is possible; and (3) to antagonise the toxins or destroy the organisms by certain antitoxins and bactericidal agents.

(1) Under this head we might enumerate all strengthening soups and easily digested articles of diet, together

with port wine and other stimulants. The patient should be fed up in most cases to more than repletion, although there are many cases of septicæmia, at least, in which overfeeding is both undesirable and impossible.

(2) Where a wound or abscess is responsible for the condition, proper surgical treatment should be enjoined, tension relieved by opening abscesses, and wounds rendered thoroughly antiseptic; although, as Watson Cheyne truly remarks, too strong antiseptic solutions are harmful, as they cause necrotic changes, and so aid rather than hinder the entrance of organisms into the body.

(3) Quinine is one of the chief remedies, in 5 grain doses every four to six hours. Whether it is really to any extent an internal antiseptic in these or any other doses, except perhaps in malaria, is open to question, but it is often of service. Sulphocarbolate of soda has been recommended, but is not of much apparent value. The antistreptococcus serum is worthy of trial in all cases, certainly where the streptococcus pyogenes is present. Life has undoubtedly been often saved through its instrumentality, and if it should do no good its careful trial will at least do no harm.

ERYSIPELAS.

Fr. *Erisipèle*. Ger. *Erysipelas* or *Pothlauf*.

DEFINITION—An infectious disease characterised by inflammation of the skin or mucous membranes, accompanied by fever and constitutional disturbances.

Erysipelas (from *Ερύω*, to draw, and *πelas*, near; or from *ερυθρός*, red, and *πελλος*, livid) is a disease characterised by fever and the appearance of a peculiar eruption on the skin. The inflammation which also attends the eruption has a tendency to spread and to invade the areolar tissue. In Scotland it is known as the *Rose*, and in England it is sometimes called *St Anthony's Fire*.

Etiology.—An erysipelatous condition may be set up by a number of organisms, but the clinical entity known as erysipelas is always caused by the streptococcus pyogenes. It was formerly thought that the streptococcus of erysipelas was a distinct species which differed from the pyogenes in certain respects, but this is now known not to be the case and the two have been definitely proved to be identical. It is true that erysipelas tends to spread from case to case as erysipelas, without giving rise to a suppurative condition, and that purulent formations caused by the streptococcus are not apt to be followed by erysipelas; but they both occasionally show this association. Thus erysipelas may be followed by a suppurative condition of the subcutaneous tissues—a cellulitis; and a true skin erysipelas may follow a purulent condition.

Morbid Anatomy.—The affected skin assumes a rosy-red colour. It is painful and swollen, the swelling being particularly great where the skin is loose, as in the eyelids. The redness spreads by contiguity and gradually dies away from the part first attacked as it spreads at the periphery, which generally shows a raised, well-defined border where it joins the healthy skin. Vesicles and bullæ may appear over the red area. Sections of this margin show dilatation of blood-vessels, leucocyte infiltration, serous exudation, and, in places, fibrin formation in the corium and subcutaneous tissues. Streptococci are most numerous in the spreading margin of the inflamed area and just beyond it, much less numerous and may even be absent in the healthy tissue further out and in the oldest and first affected parts. They lie mainly in the lymphatics, being sometimes so numerous as to completely block them, and may even cause permanent œdema. They do not usually give rise to suppurative softening of the tissues, but sometimes they spread more deeply and diffusely, and actually produce a purulent condition, which is generally known as cellulitis. Further,

they are generally restricted to the local areas of attack, however widely that may spread by contiguity, but they may escape into the system and cause inflammatory conditions elsewhere, such as meningitis, pneumonia, &c., or a septicæmic or pyæmic condition. Erysipelas most commonly occurs near a wound. In the absence of a wound, it occurs most frequently upon the face, at the junction of the skin and nasal or conjunctival or oral mucous membrane. Erysipelas has been found to sometimes exert a beneficial influence upon certain skin diseases and malignant growths, and the toxins of the streptococcus, grown along with the bacillus prodigiosus in broth and separated from the germs by filtration (Coley's fluid), are sometimes now used for injection in such cases.

Symptoms.—There is an incubation period of from one to three or four days, after which the attack generally commences suddenly with rigors and vomiting. A burning pain usually comes on in the part affected. The temperature rises rapidly to about 104°F. There is headache, thirst and the usual symptoms of the febrile state. Sore throat is not uncommon, and diarrhoea is often present. The urine is scanty and high coloured, and very frequently contains albumen and casts. So long as the skin eruption spreads the temperature keeps high, but in favourable cases both generally subside, either suddenly, which is most common, or gradually, in about a week. In unfavourable cases the high fever may continue, or delirium and great nervous prostration may set in, or severe vomiting and diarrhoea may result. There is also a much more chronic variety, in which little fever or constitutional disturbance accompany the skin eruptions, which are apt to recur from time to time, and to disappear in one place only to appear at another.

Diagnosis.—There is very rarely any difficulty except in the more chronic forms, and then a bacteriological

examination of the fluid or a scraping taken from the advancing margin of the affected skin will help to clear up all doubt.

Prognosis.—It should always be guarded in the aged or very young, especially in infants when it affects the umbilical cord, or in subjects affected with kidney disease or chronic alcoholism. Relapses occurring soon after the first attack are not uncommon.

Treatment.—The treatment consists in the adoption of measures to reduce the inflammatory action, to support the system when necessary, and to protect the inflamed parts from contact with the air. The first indication is carried out by the administration of a brisk saline purgative, and this is particularly necessary in cases of erysipelas occurring in strong and plethoric individuals. Having emptied the bowels, give the tincture of the perchloride of iron in 20 to 60 drop doses every four hours, and order the part affected to be dusted thickly over with powdered starch and boracic acid, combined in equal parts, from a dredger. Where there is a more extensive area involved, fomentations of carbolic acid (1 in 30 to 60), saturated boric lotion, or fomentations upon which liquor plumbi subacetatis has been freely sprinkled will be found of great benefit. Painting the skin round the affected area with iodine is sometimes sufficient to check the advance of the disease. The application of a mixture of the alcoholic extract of belladonna in vaseline (1 part in 3 or 4), or ichthyol ointment is most soothing. The diet during the first few days should be light and non-stimulating, consisting chiefly of milk, beef-tea, and sago or tapioca puddings. Barley water acidulated with lemon juice forms a very pleasant drink. But in the treatment of this disease, as of every other, no hard and fast rules can be laid down. Hypodermic injections of antiseptics just outside the affected area have been frequently administered

with beneficial results. Weak solutions of the perchloride or biniodide of mercury or carbolic acid are generally used in this way.

In those cases where the tincture of iron is not well borne by the stomach, quinine or the tincture of cinchona and ammonia may be substituted. A combination of the tincture of iron and the liquor ammoniæ acetatis will often be retained when the pure tincture is rejected. Wine may be given from the commencement of the attack if the patient is weak and debilitated. The sleeplessness, too often present in these cases, is best relieved by the administration of the hydrate of chloral, sulphonal, or other hypnotic.

In the phlegmonous form of the disease, deep incisions must be made into the affected part, and a large carbolic acid fomentation or a charcoal poultice applied.

Great care must be taken by doctors and nurses to avoid carrying the infection to others.

PUERPERAL FEVER.

Lat. *Febris puerperarum*. Fr. *Fièvre puerperal*. Ger. *Puerperalfieber*; *Kindbettfieber*.

DEFINITION—A continued fever, communicated by contagion, occurring in connection with child-birth, and often associated with extensive local lesions, especially of the uterine system.

The pathology of this condition is now pretty definitely known. It is merely a form of blood poisoning, showing itself as a Sapræmia, Septicæmia, or Pyæmia, in which the seat of the septic inoculation is mostly the uterus, though sometimes it is one of the other genital organs. The condition generally comes on a few days after childbirth and the local symptoms are often at first related to the genital organs. Afterwards the symptoms resemble those of other

forms of septicæmia or pyæmia. The organism which has been most frequently found, in the septicæmia forms at any rate, is the streptococcus pyogenes, but other organisms are often the causes of the condition.

Treatment.—The treatment of this disease belongs to the domain of midwifery, and to the many excellent manuals upon this department of medicine the reader is referred.

SUB-GROUP (d)—INFECTIVE DISEASES OF UNCERTAIN BACTERIOLOGY.

1.—NOT ENDEMIC.

FEBRICULA.

Lat. *Febricula*. Fr. *Fièvre éphémère*. Ger. *Febricula*.

EPHEMERAL FEVER, SIMPLE CONTINUED FEVER.

DEFINITION — Simple continued fever, non-contagious, occurring sporadically, of not more than three or four days' duration, and having no obvious distinguishing character.

Etiology.—It arises from a variety of causes, all of which are not fully known. The chief perhaps is gastro-intestinal irritation, such as is provoked by errors of diet, chills, &c., especially in children. Indefinite toxic absorption out of proportion to any local irritation which may be coincidently present may account for some of the more severe and lasting forms. Mild, ill-defined, and unrecognisable cases of some of the ordinary infectious fevers, *e.g.*, typhoid, may account for some of the instances. It is not really a separate disease; it is only the most prominent symptom in a variety of diseases.

Symptoms.—Febricula, as a rule, sets in suddenly; but in some cases, for three or four days before the fever is developed, lassitude, anorexia, and a general feeling of discomfort may exist. The febrile movement is often intense, the heat of the body rapidly rising in a few hours from the normal temperature (98.4°) to 104° F., and then as rapidly falling. The other signs of fever are present, viz., hot skin, quick pulse, furred tongue, thirst and loss of appetite, and headache. Mild delirium sometimes occurs at night, especially in children. The urine is scanty and high-coloured. The bowels are generally confined. Herpes often occurs about the mouth. The fever subsides abruptly, often with a profuse perspiration, evacuation of the bowels, or even some hæmorrhage from the nose, &c.

Diagnosis.—This rests mainly upon the sudden onset and sudden cessation of the pyrexia without the recognition of any lesion characteristic of another fever.

Treatment.—The treatment consists in emptying the stomach, by an emetic if necessary, and by the administration of saline purgatives to lessen the violence of the arterial excitement. Sodium and potassium tartrate (Rochelle Salts), 120–240 grains, in a tumblerful of lemon water or lemonade is an agreeable and efficient cathartic. The high temperature, hot dry skin, and feverish symptoms are best treated by diuretics and diluent drinks. Barley water, soda and potash waters, and cooling drinks may be taken by the patient *ad libitum*. Antipyretics, such as quinine, antipyrin, and antifebrin, are *not* indicated in the treatment of febricula, nor are they indicated in treating any simple feverish condition depending upon some passing cause. Rest in bed and a low diet are usually sufficient, and complications as they arise are treated on general principles. During convalescence, tonics and a nutritious diet must be given.

ERUPTIVE FEVERS.

The diseases now to be considered and classed under the head of Exanthemata, or Eruptive Fevers, each arise from a specific contagion, run a regular definite course, and are accompanied with a specific inflammation of the skin, called "the eruption," which also runs a definite course. The mucous membranes are likewise more or less affected.

The members of this group occur for the most part but once during life. The true exanthemata having all these characters are — small-pox, measles, and scarlet fever: there are many less perfect, as chicken-pox and the continued fevers, but in the latter the eruption is less constant and less prominent than in the true eruptive fevers. By some, these diseases have been classed under the head of Zymotic, from the theory that the poison acts in the blood like a ferment.

There are five well-marked stages in the course of these fevers:—

1. A period of incubation.
2. A stage of invasion.
3. A stage of eruption.
4. A stage of desiccation.
5. A stage of convalescence.

TABLE giving the Appearance of the Characteristic Rashes in the following Diseases.

SCARLET FEVER.	SMALL-POX.	TYPHOID.	TYPHUS.	MEASLES.
<p>A crimson rash composed of minute slightly elevated dots. First seen on the second day of the fever over lower part of abdomen, inner sides of thighs, and then over rest of body. Body colour of <i>boiled lobster</i>. Desquamation follows.</p>	<p>It appears first on face and backs of wrists during third day of the fever as distinct papule, elevated, acuminated, of a florid red colour, and giving a peculiar "shotty" feel to the finger. The papules become vesicular and then pustular, and lastly a scab is formed.</p>	<p>It appears about the seventh day on chest and abdomen. It comes out in successive crops of rose-coloured, slightly elevated, lenticular spots or papules, in size somewhat less than a split pea. The rash disappears on pressure. Each crop remains visible for three or four days, disappears and then reappears on another part of the body.</p>	<p>It appears between third and seventh day of fever. A mulberry coloured rash all over the body, seldom on face, imperfectly disappearing on pressure, and continuing till end of fever. May become petechial.</p>	<p>The eruption appears on the fourth day as dingy red-coloured papules on the face, close to the roots of the hair, slightly raised above the surface, and having a tendency to form crescentic patches.</p>

TABLE showing Points of Distinction between Small-Pox, Measles, Scarlet Fever, and Chicken-Pox.

	SMALL-POX.	MEASLES.	SCARLET FEVER.	CHICKEN-POX.
<i>Period of Incubation....</i>	Nine to fifteen days, often twelve	Ten to fourteen days.	Two to six days.	Ten to fifteen days.
<i>Prenomitory Symptoms..</i>	Pains in the limbs, and especially in the <i>back</i> . Sudden chills and rigors.	Those of a catarrhal attack.	Chills, rigors, and sore throat, intense heat of skin.	Slight feverishness and malaise.
<i>Eruption appears.....</i>	Third day of fever as a papule with a shotty feel, then vesicular, and lastly pustular. First on face, especially on forehead, back of the wrists, and then spreads over body.	Fourth day of fever. First on face close to the hair, and then spreads over body in crescentic patches.	Second day of fever. First on neck and upper part of chest and inner sides of the thighs, then spreads over face and body.	In some cases, after two or three days' fever. First on shoulders and chest. Few spots on face. Most frequently there is no preliminary fever.
<i>Eruption fades</i>	Scabs form on ninth or tenth day of fever, and fall off about the fourteenth.	Seventh day of fever.	Fifth day of fever.	The contents of the vesicles become cloudy on the second day, and dry upon the fourth.
<i>Character of Eruption...</i>	At first a distinct papule, elevated, acuminate, of a florid red colour, and giving a peculiar "shotty" feel to the finger. The papules become vesicular and then pustular, and lastly a scab is formed.	Papules of a dingy red colour, but slightly elevated, and having a tendency to form crescentic patches. To the finger they give the sensation of being passed over a well-worn nutmeg grater.	A crimson rash composed of minute, slightly elevated dots. The rash extends over the whole of the body, which has the appearance of a boiled lobster. Desquamation follows.	Small red spots which in a few hours become limpid vesicles about the size of a pin. The surface of the body looks as if sprinkled with water. The contents of the vesicles become cloudy but never purulent. The vesicles dry up and the crusts fall off, seldom leaving any cicatrix.

VARIOLA—SMALL-POX.

Lat *Variola*. Fr. *Variole*. Ger. *Blattern*. Syn. *Menschenpocken*.

DEFINITION—A contagious and infectious continued fever, resulting from the absorption of a specific poison, and characterised by the appearance of a peculiar eruption, commencing on the third day of the fever.

Small-pox is the type of the zymotic class of diseases and was first described by Rhazes, an Arabian physician and botanist. Natural or unmodified small-pox is now a rare disease, owing to the practice of vaccination. It prevails chiefly in winter and spring and appears to have a tendency to recur in its epidemic form once in ten years. Inoculation, by which the practice of transplanting the virus of small-pox from infected to healthy persons is known, was practised in China from the 6th century, but it was not till Lady Mary Wortley Montagu wrote her celebrated letter from Adrianople in 1717 that the operation became generally known in England. Caroline, Princess of Wales, had two of her children inoculated. The practice was opposed by the clerical and medical professions, a member of the former making the discovery that "Job's distemper was confluent small-pox, and that he had been inoculated by the devil." The objection of the medical profession was more to the point in that the practice of inoculation tends to propagate the disease. Since the introduction of vaccination, inoculation has been made illegal.

Small-pox has been divided into two varieties—*variola discreta* and *variola confluens*. In the first variety all the pustules are *separate* and *distinct*; in the second they coalesce over the greater part of the body. There are also varieties known as *corymbosa*, when the eruption is arranged in clusters; *malignant*, *hemorrhagic*, *benigna*, when the pocks dry up without becoming purulent; and *variola sine eruptione*.

Confluent small-pox has a tendency to become malignant, a form of the disease which when once seen is not likely to be forgotten.

There are four well-marked stages :—

1. A period of incubation of from twelve to fourteen days' duration. When the disease has been inoculated this period is from seven to eight days.
2. A stage of invasion. Two to four days.
3. A stage of eruption (beginning on the third day).

{	(1) Eruption about five days.
{	(2) Suppuration four or five days.
4. A stage of desiccation (from ninth to tenth or eleventh day).

}	Six to ten days.
---	------------------

Symptoms.—After a period of incubation an attack of small-pox is generally ushered in by a sensation of chilliness and rigors alternating with flushes of heat. Nausea followed by vomiting is often an early symptom. The tongue becomes furred, the appetite lost, the throat sore, and pain is complained of in the epigastric region. Cephalalgia, pains in the limbs, *back*, and loins are also present. At this stage the disease may be mistaken for a severe bilious and catarrhal attack. Pains in the loins with headache, vomiting and constipation are important diagnostic symptoms, and so also are the frequent perspirations. The absence of these perspirations if accompanied with diarrhœa in adults is said to betoken a severe attack. Children are often seized with convulsions in this early stage. On the second day of the disease the thermometer may mark 105°F. of heat (*primary fever*); the temperature then falls on the appearance of the eruption, but during the period of suppuration (*secondary fever*) it again rises to 102°

or 105°F., the evening temperature being somewhat higher than the morning. This irregular course of rise and fall continues till about the eighteenth day, when the normal temperature is reached. A peculiar disagreeable smell rises from the patient during maturation and bursting of the pustules. In the confluent form of the disease, to the above is added greater severity; the pain in the loins is greater; the tonsils and parotid glands are inflamed and swollen; the eyelids swollen, and the eyes closed; the lips are puffy, and a constant flow of saliva dribbles from the corners of the mouth; the urine becomes scanty, but never suppressed, and charged with urea and uric acid, albumen for a few days is sometimes present, and even blood in severe cases may appear in the urine. The pulse is feeble and quick, and symptoms of increasing debility make their appearance. This secondary fever, usually slight in the distinct variety, becomes intensified in the confluent, and the patient may die suddenly, unable to contend against the virulence of the attack. Delirium in these cases is of frequent occurrence, and the danger to life becomes great, the most fatal period being between the eighth and thirteenth days, but especially the eleventh.

Description of the Eruption.—The eruption runs a definite course, appearing first on the forehead, neck, and backs of the wrists in small isolated inflamed points, called *papulæ*; these, extending thence over the body and lower extremities, on the fifth day become *vesicular*, the cuticle being raised and filled with serum. On the seventh day each vesicle becomes umbilicated by the appearance of a central depression. On the eighth day the vesicles suppurate and become *pustular*, the central depression is lost, so that the pustules are orbicular, and are surrounded by a bluish inflamed ring, termed an *areola*. About the eleventh day the process of scabbing takes place, the pustules bursting and then gradually drying up. The fever, as soon as

the eruption appears, *remits*, but returns during the suppurative stage of the pustules. This return of the fever (*secondary fever*) is considered by some to be due to the specific nature of the disease, by others to result from the process of maturation in the pustules, and to partake somewhat of the nature of a *suppurative fever symptomatic* of the local affection. There is a well-marked leucocytosis during the stage of suppuration. Besides the eruption above described Dr Fagge describes two other forms of eruption occurring during the course of small-pox, but chiefly in its initial stage—the *roseola variolosa*, one form of which is maculated and is more or less like measles, coming out on the second or third day on the face and then extending over the body; the other variety is an evenly diffused red bluish, resembling scarlet fever. Its presence as a triangular patch with its base at the umbilicus and apex at the pubis enabled Dr Fagge “to diagnose correctly a case in which there was no other reason to suspect small-pox.”

Complications, &c.—Besides the effects of the small-pox poison on the system—the fever and the eruption—there are others which, for the sake of convenience of description, may be tabulated as follows:—

1. Vascular and Respiratory Systems.
2. Genito-Urinary System.
3. Cutaneous System.
4. Digestive System.
5. Nervous System.

1. *Vascular System*.—Myocarditis sometimes occurs, but other cardiac complications are rare. Lobular pneumonia is common, and pleurisy also in some epidemics, but other complications are rare.

2. *Genito-Urinary System*.—Albuminuria is common but kidney disease rare. Orchitis and ovaritis sometimes occur, and a pregnant woman may abort.

3. *Cutaneous System.*—The areolar tissue under the skin is in some cases the seat of numerous small abscesses, which form in rapid succession on the limbs. These abscesses seldom contain healthy pus. The cuticle is frequently softened, and has a marked tendency to peel off. In confluent small-pox the vesicles on the body and extremities, though often not confluent, are pale, and have no areola, and the face is pale and doughy. The vesicles have by careful dissection been dissected out, and it has been found possible to puncture the vesicular portion of the eruption and let out its contents without disturbing the pustular portion, which may be also treated in a like manner; the eruption, in fact, consisting of a vesicle surrounded by pus, placed in a cup-shaped cavity. The pitting of small-pox is the result of the extension of the pustular affection into the true skin, or *rete mucosum*, and the destruction of the part thus attacked.

4. *Digestive System.*—Whether the pustular eruption of small-pox is ever present on the mucous membrane of the stomach and intestines may admit of doubt. It may, however, occur in the mouth and on the fauces, giving rise to the sore throat so often complained of by the patient. The probable explanation of their supposed presence on the intestinal mucous membrane may be that suggested by Aitken, who remarks that “the appearance of eruption in such cases is due to the solitary mucous glands, which are filled with exudation, not of a purulent kind, but having all the external appearance of pustules.”

5. *Nervous System.*—They are important and often serious. Convulsions occur in children and sometimes mania in adults. Fatal coma may follow delirium.

The *sequelæ* are—deafness (due to suppuration of the internal ear), blindness, abscesses, boils, &c.

With regard to the affection of the eye, often ending in blindness, the late Mr Marson says:—“The eye seems to possess a complete immunity from the small-pox eruption,

and that although it sometimes extends to the inner margins of the eyelids, the particular local affection that causes the destruction of the organ of vision in variola begins generally on the eleventh or twelfth day, or later, from the first appearance of the eruption, and when the pustules in every other part of the body are subsiding. It comes on after the secondary fever has commenced, with redness and slight pain in the part affected, and very soon an ulcer is formed, having its seat almost invariably at the margin of the cornea. This continues to spread with more or less rapidity, and the ulceration passes through the different layers of the cornea until the aqueous humor escapes, or till the iris protrudes. In the worst cases there is usually hypopion, and when the matter is discharged the crystalline lens and vitreous humor escape. In some instances the ulceration proceeds very rapidly. I have more than once seen the entire cornea swept away within forty-eight hours from the apparent commencement of the ulceration; and, what is singular, now and then the mischief goes on without the least pain to the patient, or his being aware that anything is amiss with his eyes."

Diagnosis and Prognosis.—The diagnosis prior to the stage of eruption cannot be made with certainty. In measles the stage of invasion is longer, and is marked by catarrhal symptoms; in scarlet fever the rash appears on the second day, and the throat is early affected (see Table, page 148). Small-pox, as a rule, occurs but once in the same person, but cases are on record of two, three, or even more attacks in the same individual. It may also be combined with measles, whooping-cough, ague, &c. In these cases it is more or less modified by the concurrent disease. The fatality in natural or unmodified small-pox is great, but the danger to life is greater in some epidemics than in others. The confluent variety is more fatal than the discrete, the danger in the former being greater during the

stage of the secondary fever. The eleventh day of the fever, or the eighth day of the eruption, is the period at which death most frequently occurs. Death is generally due to a combination of asthenia and coma. The danger to life is greater among children than adults. Pregnant women are said, as a rule, to abort and die, but exceptions not infrequently occur.

Dr Watson says:—"There is no contagion so strong and sure as that of small-pox: none that operates at so great a distance." Unequivocal evidence of minute scales of small-pox matter have been detected by Bakewell in small-pox wards, and these may be the means of propagating the disease. Small-pox is more contagious in its later than earlier stages. No specific organism has yet been discovered.

Treatment.—The first duty of the medical man is to isolate the patient, and the next to prevent the spread of the disease by vaccinating every one who has been in contact. It is rare, indeed, to find that a small-pox patient has a second attack, and vaccinia protects much in the same way as an attack of the disease itself, and therefore vaccination, or rather revaccination, every seven years ought to be absolutely compulsory, and especially when an epidemic of small-pox is imminent.

The patient attacked with the disease should be placed in a small-pox hospital, or, if that is unattainable, in an isolated room, which should be large, airy and well ventilated, but kept at a temperature of 50° to 60°F.

All carpets, curtains and superfluous furniture should be removed, and the bed selected should be of small size, so as to facilitate nursing. The bed-clothes should be light, but warm, and the night-dress and sheets should be of fine linen, so as to obviate irritation of the skin.

The window should have red or orange glass, or failing that, red tissue paper may be pasted on, or red curtains

used. This precaution against the more actinic violet rays of light prevents scarring to a considerable extent. Careful nursing is of the utmost moment, and the strictest attention to cleanliness is necessary, especially where purulent discharge from the pustules may soak into night-shirts and sheets. Perhaps it may be more truly asserted of this disease than of any other that a good nurse is of much more importance in a bad case than a skilled physician.

The diet, which at first will probably be simply milk with aerated water, may be increased in the vesicular stage to beef tea, chicken soup or jelly, custards and so forth, and in the pustular stage the patient requires food frequently and is often much the better of some stimulant. During this stage and when the pustules have burst there is often considerable absorption of toxic matters, and this is best treated by food and a sufficiency of alcohol, the quantity depending much on the patient's age, habits and strength. Occasionally peptonised food is necessary, and during convalescence the appetite may be stimulated by easily digested but well seasoned and cooked dishes. Thirst may be satisfied with plenty of water.

Sponging the skin is of great value, and there is distinct advantage in using a weak antiseptic lotion, such as boracic acid, permanganate of potash (which, however, stains the skin), carbolic lotion or thymol.

The eyes require constant attention, especially in confluent cases, as pocks frequently form on the conjunctiva and may lead to deep-seated ulceration and perforation. When pocks appear on the eyes, or in the nose or mouth, washing with boracic lotion must be carefully enjoined.

Delirium when present taxes the nurse's skill, but much may be done by humouring a patient where physical restraint would be most prejudicial.

Drugs internally may not be required at all. Sometimes, however, opium, chloral, or bromides may be given to secure sleep and relieve the excessive discomfort and

irritability of the skin during maturation, and these drugs are invaluable when delirium threatens.

Constipation, which is often present, should be treated by saline cathartics, and diarrhœa when it appears as a result, sometimes of septic absorption, should be treated on the usual lines.

More important is the treatment of the skin. Cold water compresses are most soothing and salve muslins of ichthyol and oxide of zinc may be applied to any part of the skin which is unduly irritable. Some authorities recommend pricking any tense vesicles or pustules which are specially irritating the patient. This procedure is often of great value. Linseed poultices have been recommended for loosening the crusts, and various antiseptics in vaseline and lanoline are most serviceable as well as soothing. Tepid baths aid crusts to form and help in their removal, besides giving great relief to the patient.

Should deep-seated inflammations occur they must be treated with the usual surgical measures, and when pus forms freely opened. Complications should be treated as they arise, but especial care must be expended on the eyes if they become inflamed. Atropin instillation is of value if a corneal ulcer appears. During convalescence much may be done by massage to restore the skin to its normal appearance.

VARIOLOID.

Under this head are now classed such diseases as were formerly known and described as *stone-pock*, *horn-pock*, *wart-pock*, but which are now considered to be small-pox more or less modified by vaccination.

The following list of differences between small-pox and varioloid, given by Aitkèn, will help to distinguish the two diseases :—

1. A fever of three days, without eruption, affecting people during variolous epidemics.

2. A high and severe fever, followed by a mild eruption, sometimes only a single pock ; the slight proportion which the amount of eruption bears to the severity of the preceding fever is, perhaps, the most marked characteristic of varioloid.

3. The occasional appearance of a scarlet efflorescence, like that of a scarlatina or roseola, preceding the appearance of the proper pimples, which occur as a scanty crop.

4. In some rare instances the eruption is confluent, but does not advance beyond the development of a pimple or vesicle, and begins to dry on the fourth or fifth day of the eruption, forming a small hard tubercle, which soon disappears.

5. Sometimes the eruption is pimple, vesicle, and pustule at one time in the same case.

6. Sometimes the eruption runs its regular course, but stops sooner—on the sixth or seventh day, instead of the eighth or ninth. In general, it may be stated that the severity and fully-developed state of the disease is in proportion to the length of time which elapses from vaccination.

7. The varioloid eruption wants the peculiar odour of natural small-pox, and secondary fever is very rare.

8. Other eruptive affections, such as measles, scarlatina, and purpura materially modify the course and symptoms of small-pox.

Treatment.—As in mild forms of small-pox.

VACCINIA—COW-POX.

Lat. *Vaccinia*. Fl. *Vaccine*. Ger. *Kuhpocken*.

DEFINITION—A contagious disease transmitted from the cow to man by vaccination or inoculation.

To Dr Jenner is due the discovery that cow-pox communicated to man renders him less susceptible to the contagion of small pox. The generally received, and probably the only correct, opinion is that vaccinia, or cow-pox, and variola are identical, the poison of the latter being modified and rendered milder by passing through the system of the cow. Dr Moncton Copeman has shown that when small-pox is inoculated into a calf a disease is produced which although it has lost the infectiousness of small-pox is still capable of affording protection from that disease, as vaccination does against subsequent vaccination or variolation. Pure calf lymph is now largely used, and is free from the objection that vaccination is a propagation of syphilis and tuberculosis. Drs Copeman, Crookshank, and others have further shown that vaccine lymph, whether stored in the liquid state or freshly obtained from a normal vesicle, contains micro-organisms of various kinds, for the most part mere saprophytes. "These," says Dr Copeman, "along with chance of super-addition during or after vaccination of pathogenic organisms by agency of careless people, whether vaccinators or persons having the charge of infants, have been advanced as reasons for doing away with the practice of vaccination, on the grounds of the possible harmfulness of the extraneous organisms liable to be introduced into the system at the time or in the course of vaccination." This objection may be entirely set aside by mixing a certain amount of chemically pure glycerine with the lymph and storing the mixture for a time before it is used. By this process the ordinary saprophytes,

together with the streptococcus of erysipelas, are destroyed, while the activity of the lymph is not impaired, but even improved.

In view of the constant controversy on the merits of vaccination the student should be prepared to meet the following objections. That vaccination is not a prophylactic to small-pox. This disappointment is due to—

1. Absence of full statistics showing the mortality of the vaccinated compared with that of the unvaccinated.
2. Medical men not stating in their death certificate if patients unvaccinated or marks present if vaccinated.
3. Vaccination improperly performed, from carelessness on the part of the operator, or in deference to the wishes of the parents to have as few marks as possible.
4. The relative number of vaccinated and unvaccinated to the entire population.

Two positive statements may be made since the introduction of vaccination—

1. That the mortality from small-pox has greatly lessened.
2. That the fearful disfigurement once so dreaded and so characteristic is now almost unknown among the vaccinated.

There are several methods by which the operation of *vaccination* may be performed, but the following may be taken as a guide. The usual place chosen for implanting the lymph of cow-pox is the skin covering the deltoid muscle of the arm. The arm is grasped by the left hand of

the operator, and the skin being tightened, a clean sharp lancet, charged with lymph, is passed gently into the skin, so that a valvular puncture is made, and the vaccine virus allowed to enter the wound.

If the operation be successfully performed, about the second day small red points appear raised over the sites of the punctures. Papules on the fourth day become developed, and on the fifth or sixth day distinct vesicles are discoverable. On the eighth day they attain to their full development, and are surrounded by an areola. On this day also the vesicle is distended by a clear lymph. About this time there is usually some slight fever, the lymphatics of the arm are swollen, and the axillary glands enlarged. Soon after the eighth day the contents of the vesicles become more or less purulent, and a black spot is seen in the centre of the pock. On the fifteenth day a hard blackish scab is formed, which generally falls off about the twenty-fifth day from the date of the operation. A lichenous eruption often attends or follows vaccination. The best age at which to perform the operation is before the child is three months old, and before the process of dentition has set in. The child should also be in the enjoyment of sound health. On re-vaccination, if the patient is susceptible, the local effects appear earlier than in the primary vaccination, and the local and constitutional symptoms are more severe.

Treatment.—Saline aperients and the application of a charcoal poultice, when necessary, to the inflamed pocks should be sufficient treatment where due care and aseptic precautions have been taken.

VARICELLA—CHICKEN-POX.

Lat. *Varicella*. Fr. *Varicelle*. Ger. *Windpocken*. Syn. *Wasserpocken*; *Varicellen*.

DEFINITION—A contagious disease, occurring chiefly in early life, attended with a vesicular eruption and slight fever.

Chicken-pox requires no special treatment beyond the restriction of the diet and the administration of saline aperients. The vesicles usually dry up between the fourth and seventh day of the disorder, leaving small red spots, but seldom cicatrices on the skin. It is of importance to early distinguish this disease from variola, so as to allay the anxiety of the patient's friends.

The following description may help the student to distinguish small-pox from chicken-pox:—

In small-pox the premonitory symptoms are well marked—rigors, considerable fever, and severe pain in the loins.

In chicken-pox, on the other hand, the fever, if present, is slight, and the patient—most frequently a child—scarcely complains of feeling ill; “the body in varicella has the appearance of having been exposed to a momentary shower of boiling water, each drop of which had occasioned a minute blister.” The vesicles do not become pustular unless irritated. The pocks come out in crops, rarely appear on palms of hands or soles of feet, and in all may number 200.

In some cases the disease is so severe that it cannot easily be diagnosed from small-pox. The diagnosis will be assisted by noticing that the severe symptoms seldom last twelve hours; thus the child would appear very ill at night and almost well the next morning. The eruption may also be confluent in some cases. The rapidity of recovery, the absence of pitting, and freedom from the complications attendant on small-pox, will help the diagnosis.

Treatment.—A mild saline aperient and, where necessary, an ordinary febrifuge mixture should be administered. Scratching must not be permitted, and a soothing lotion or ointment often allays the itching, which, in severe cases, may almost equal that experienced in small-pox. For this, similar treatment to that recommended in small-pox should be used.

SCARLATINA—SCARLET FEVER.

Lat. *Febris rubra*. Fr. *Scarlatina*. Ger. *Scharlachfeber*.

DEFINITION—A contagious and infectious fever, characterised by a high temperature: sore throat and a scarlet eruption appearing on the second day of the fever, and followed with desquamation of the cuticle.

Scarlet fever is the type of an infectious malady, and recent investigations have suggested that it may be a disease of cows transmitted to the human-subject. The disease in cows which is supposed to give rise to scarlet fever occurs as small vesicles and ulcers on the udder and teats. This is known as the "Hendon Disease." The streptococcus of scarlet fever (Klein) was found in them, and a sub-culture of these from human scarlet fever given to the calves produced the Hendon disease. The streptococcus conglomeratus of Klein is now largely discountenanced as the cause of scarlet fever. Class has recently described a diplococcus conglomeratus which he has obtained from the throat, blood, and scales of scarlet fever cases. He states that it is invariably present. The question is still an open one. The poison appears to be less volatile than measles, but acts at a greater distance than typhus. It appears to be now generally accepted that the chief means of

propagating scarlet fever are by the secretions of the throat, nose, and ears. It may be propagated in every possible way, even by letters sent by the post. Healthy persons may convey the poison without being themselves attacked. Schools frequently spread the disease. Scarlet fever is most prevalent in autumn, the mortality being highest in October and November. The death-rate is highest among young children, that is, under five years of age, adding about 0·5 per 1000 to the death-rate. Scarlet fever may disappear for three or four years and then reappear, due probably to the fact that the liability to the disease during the first year is almost *nil*, that it increases up to the fifth year and then declines. For a few years after an epidemic the community is thus to a great measure protected. More females are attacked, but more males die. The susceptibility to the disease diminishes after the fifth year. Till the time of Sydenham scarlet fever and measles were confounded together.

There are three varieties of scarlet fever:—

SCARLATINA SIMPLEX.—*Definition*—A scarlet rash, with redness of the throat, but without ulceration.

SCARLATINA ANGINOSA.—*Definition*—A more severe form of the disease, with redness and ulceration of the throat, and a tendency to the formation of abscess in the neck.

SCARLATINA MALIGNA.—*Definition*—The throat tends to slough; the scarlet rash is scarcely, if at all, visible. Petechiæ are often seen on the surface, and the fever is of a low form.

Symptoms.—After a period of incubation varying from a few minutes to four or five days, seldom exceeding six (Murchison), scarlet fever sets in suddenly as a rule, with a sensation of chilliness, rigors, vomiting, and *sore throat*,

the two last being early and prominent symptoms. In children more frequently than in adults vomiting or convulsions may be the first symptoms ushering in an attack of scarlet fever. The temperature rapidly and steadily rises to 104° or 105° and attains a greater elevation in this than in the other eruptive fevers, and, unlike small-pox, does not abate at the appearance of the eruption. The pulse may also rise in children to 140 or 160 in a few hours, but this rise does not indicate any severe complication. If the throat be early examined, redness of the fauces, more or less vivid, may be observed, and there is some pain experienced during an attempt to swallow. Hæmorrhage from the nose in this stage is not uncommon.

This is the stage of invasion, the scarlet eruption appearing on the second day of the fever. The eruption is generally first noticed on the body and limbs before it makes its appearance on the face. It occurs primarily in the form of small red dots, more or less raised above the surface of the skin. These dots soon coalesce, forming irregular patches, varying in size and shape. The redness is scarlet, of a boiled lobster colour as described by Watson, and is brightest at the bends of the joints, *inner parts of the thigh, and the lower part of the abdomen*, but is not so uniform as in the case of erysipelas, and disappears temporarily on pressure—the reappearance being from without inwards, that is, towards the point where direct pressure was made. Some observers have likened the eruption to minute dots of red ink on blotting-paper. The maximum of diffusion and intensity of the rash is reached on the third day after its first appearance; after that period it begins to fade. In some cases, more frequently in the adult than in the child, the rash is altogether wanting, the only signs of the disease present being the continued high fever and sore throat. These cases are not infrequently followed by albuminuria, leading one to refer the previous suspicious attack to its right source—scarlet fever.

Mild cases may escape observation. Convulsions may be the first important signs, and in such cases a careful examination may show desquamation, and a rigid cross-examination may elicit that the child has suffered from a slight sore throat and rash which the parents did not think "amounted to much." Such mild attacks are often followed by the most severe sequelæ.

In scarlet fever the temperature on the evening of the second day is about 105.6°F . It may then rise on the third day to 105.8° and from that day till about the ninth day it vacillates between 103.8°F . and 102.9°F . On the tenth day it falls to 100.9°F ., and then defervescence continues uninterruptedly till the normal temperature is reached—about the fifteenth day. Dr Sydney Ringer has shown that the temperature in scarlet fever falls on the fifth day of the disease, then on the tenth, and so on, each fall being separated by an interval of five days. Compare this course in the temperature of this disease with that of measles; the sudden fall from 105°F . on the fifth day to 102°F . on the seventh day is typical of the latter disease.

The throat in mild cases of scarlet fever is not very sore, some patients scarcely complaining of any inconvenience. In *scarlatina anginosa* and *maligna*, on the other hand, the affection of the throat is always a prominent and distressing symptom. The glands of the neck become secondarily affected, inflamed and painful. The appearance of the tongue is peculiar; at first it is greatly furred; the papillæ, projecting through the coating, give it the appearance of having been sprinkled with red sand. As the disease progresses the coating peels off, leaving the surface of the tongue clean and red, the enlarged papillæ giving to it a *strawberry-like* appearance, especially at the tip and edges.

During the eruptive stages delirium generally exists, often requiring the attendance of a watcher to prevent the patient from injuring himself. Slight albuminuria is often

present. The duration of the stage of eruption is from four to six days.

Desquamation usually sets in prior to the entire disappearance of the rash. At first the cuticle comes off in small scales like scurf, but it can at times be removed in small sheets, especially from the hands and feet. Sometimes even the nails are exfoliated, and even when this does not occur, transverse grooves may often be seen on them, showing an arrest of their growth and pointing to an explanation for the origin of sequelæ, which otherwise would have remained unexplained. These grooves in time disappear, and although they are not peculiar to scarlet fever, their presence is, to say the least, suspicious. Troublesome pruritus, often intense, accompanies this stage. Albuminuria is also present, but, unless in a very large amount, does not indicate any alarming complication.

Not infrequently, during the course of scarlet fever, the patient complains of severe pains in the joints, of a rheumatic character, which often prevent his sleeping.

So far, we have only described a mild case of scarlet fever; but others, unfortunately, too frequently occur, when the disease assumes a most malignant type. In *scarlatina anginosa* and *maligna* the whole force of the poison seems to be directed to the throat and cervical glands—the latter becoming so enlarged as to form a great lump on both sides of the neck. The tonsils are enlarged so as almost to block up the entrance to the pharynx, and are covered by a membranous exudation. Necrosis occurs in the tissues of the throat, and the fetor of the breath is extreme. The act of deglutition is performed with difficulty, often with great suffering. The rash is slight, sometimes absent. The severity of the throat affection often leads to a rapidly fatal termination, resembling in this way malignant diphtheria, death, in some cases recorded by Dr Kennedy of Dublin, taking place in two days from the commencement of the disease. In cases where the throat symptoms are

prominent, it is to be remembered that diphtheria as well as scarlet fever may be present, and a bacteriological examination must be made. Towards the fatal termination diarrhoea may set in, or the child may appear to have taken a turn for the better, swallowed some milk or beef-tea with eagerness, and then died in the course of three or four hours.

The *sequelæ* of scarlet fever are albuminuria and dropsy, generally coming on during the stage of desquamation if care has not been taken to protect the patient from cold. An attack of dropsy may be the first intimation that a patient had been through a mild and unrecognised attack of scarlet fever. The earlier the nephritis develops in the course of the disease, the more intense it is. It is most common in the second or third week. Deafness is not an infrequent sequel, and may be due to perforation of the membrana tympani, to closure of the Eustachian tubes, or to disorganisation of the internal ear by the extension of the inflammation from the throat. Inflammatory brain mischief, *e.g.*, meningitis, may subsequently follow upon the otitis.

When dropsy occurs, oedema is generally first noticed in the face and lower extremities.

Convulsions and coma, due to uræmic poisoning, may also occur as sequels to scarlatina.

Diagnosis and Prognosis. — The early diagnosis of scarlet fever is by no means easy, and the student will often be sorely troubled to decide as to the nature of the case before him. During an epidemic of any of the eruptive fevers, we not infrequently meet with cases presenting symptoms more or less identical with those of the prevailing epidemic, which, however, pass over, and prove by their termination to be perfectly harmless. During the epidemic of small-pox (1876–1877), many cases of what appeared to be at first sight scarlet fever occurred. There

were present slight fever, sore throat, and an eruption closely resembling that of scarlet fever. All these symptoms passed off in a few days, and the patients, mostly children, appeared in their usual health. These cases may be, as some suppose, mild forms of scarlet fever; at any rate, they greatly embarrass the medical man. Persons may walk about with the small-pox, apparently unconscious of the nature of their illness. From what has been said, however, the practitioner will, we hope, be on his guard, and be careful before he offers too hasty an opinion; and he had better err on the side of too much caution than fall into the mistake of over-precipitation in pronouncing the disease harmless, and thus risking the lives of other members of the family to whose assistance he has been summoned. Acute exfoliative dermatitis also closely resembles scarlet fever. Measles and diphtheria are also sometimes mistaken for it in their early stages. A well marked leucocytosis is a good diagnostic guide in scarlet fever.

The *Prognosis* must be guided by the nature of the symptoms and the severity of the throat affection. Convulsions and coma are often the forerunners of a fatal termination. Death may sometimes be due to the formation of a clot in the right cavities of the heart.

Scarlet fever is very infectious. In spite of the greatest care to properly cleanse and disinfect houses, scarlet fever may again break out in the same building after the lapse of some months. "You will be asked," says the late Sir Thomas Watson, "at which period the danger of imparting the disease on the one hand or of catching it on the other is over; and I would recommend you to answer that you do not know. I am sure I do not, and therefore I always decline the responsibility of giving an oracular opinion on the matter." One attack is generally a preventive of a second, but the exceptions to this rule are by no means rare.

Among the infectious diseases scarlet fever ranks second in its fatality, being, however, more fatal in towns than in the country.

Treatment.—In simple cases, little is necessary by way of treatment beyond the usual measures adopted in fevers, viz., good ventilation and the prevention of exposure to cold. Cooling drinks to allay thirst, and simple nourishment should be ordered. Ice can be freely given. The bowels should be relieved if necessary by the administration of saline aperients or enemata. Sydenham maintained that scarlet fever was only dangerous from the officiousness of the doctor.

There is no specific for the cure of scarlet fever; sound common sense, united with careful watching of the case, so as to be ready to meet every untoward symptom as it arises, is superior to all reputed specifics. The affection of the throat is best treated by a gargle of the chlorate of potash, permanganate of potash, or a weak solution of chlorinated soda; while spraying or washing out mouth and nose with sulphurous acid, carbolic acid and glycerine or other antiseptic solution of suitable strength is of much value. In the case of young children the lozenge of the chlorate of potash (B.P.) may be sucked along with a black-currant lozenge; and in this way we have succeeded in disguising the taste of the chlorate of potash, and cheating our young patients. For hyperpyrexia the application of the wet sheet, or *wet pack*, is of much service. Sponging the body with tepid water may often afford great relief and help to diminish the intense heat of the skin. Cold compresses should be applied to the throat if the patient will submit to them. Of course, in the malignant forms of the disease alcohol must be given; quinine and the chlorate of potash or carbonate of ammonia will form the chief medicines from the administration of which any hope may be derived. The dropsy where renal disease develops must be treated by

the exhibition of the tincture of iron, and by free purgation with elaterium or jalap. Dry cupping over the loins or hot fomentations to the same part are of great use when the kidneys become affected. The daily examination of the urine for albumen should be routine practice, even where there is no suspicion of acute catarrhal nephritis. Extreme restlessness may require the administration of anodynes; hyoscyamus and belladonna are to be preferred to opium. During the period of desquamation the patient may be well rubbed with weak carbolic oil (2-5%), carbolated vaseline, or thymol in vaseline (10 grains to the ounce), and then placed in a warm bath, care being taken to prevent cold. The utmost precaution must be taken to prevent involvement of the middle ear. This is partly guarded against by careful antisepsis of the orifices of the Eustachian tubes, but if, notwithstanding every care, the middle ear becomes involved, then the tympanum must be examined daily so as to permit of lancing the membrane should excessive bulging occur. The operation can be carried out painlessly under the influence of cocain applied locally before puncture. Afterwards the ear should be kept carefully washed out and plugged lightly with antiseptic wadding. Puncturing the drum may require to be done several times over, where fluid continues to gather in excess in the middle ear. Lastly, be careful in your recourse to alcohol, for by beginning its administration too soon, you rob yourself of a valuable assistant in the hour of need.

MORBILLI, RUBEOLA, OR MEASLES.

Lat. *Morbilli*. Fr. *Rougeole*. Ger. *Masern*.

DEFINITION—An acute highly infectious continued fever, commencing with well-marked catarrhal symptoms, the presence of a mulberry-coloured rash appearing on the fourth day of the fever and disappearing on the seventh.

Etiology.—The disease can be communicated by inoculation and by fomites, and is far more infectious than either small-pox or scarlet fever. It spreads more rapidly than the diseases just mentioned, owing probably to the fact that its infectiousness is early developed, and that it is about the second day of its invasion before the true nature of the attack can be fully recognised. Dr Koplik of New York has described an eruption on the inside of the lips and buccal mucous membrane which he says appears *before* the eruption on the skin and is pathognomonic of measles. The eruption “consists of small irregular spots of a bright red colour. In the centre of each spot there is noted, in strong daylight, a minute bluish-white speck.” The spots of simple aphtha, accompanied by febrile movement, are not as bright red as the spots of measles, and they do not show the bluish-white spots above described. The infection is communicated by the breath and secretions, especially the nasal. It is also conveyed by fomites. The specific organism has not yet been discovered, and though many claims have been advanced, none has yet been established.

Children are much more frequently attacked than adults; but it has been known to prevail as an epidemic among troops. Adults are more frequently attacked than in scarlet fever. The mortality at all ages is about 0.4 per 1000, but under five years it amounts to 2.8 per 1000 living. The mortality is probably due to intercurrent capillary bronchitis, which is a most fatal disease among

young children, for measles itself rarely kills. Though comparatively a mild disease in Britain, measles when introduced to a new land is most fatal. It nearly depopulated the South Sea Islands, as small-pox did the Central and Northern parts of North America. This severity is supposed to be due to the absence of immunity established by previous attacks. As a rule measles occurs but once during life, but there may be a second, third or even fourth attack.

Symptoms.—Measles, unlike the other eruptive fevers, sets in with *well-marked catarrhal symptoms*. The eyes water, the nose runs, and the patient presents all the appearances of suffering from a severe cold.

The period of incubation of measles is from ten to fourteen days, *the rash appearing on the fourth day of the fever* and fading away on the seventh. The eruption first appears on the forehead and temples, close to the roots of the hair, extending in a few hours over the body and limbs. It is of a dull-red or mulberry colour, occurring in the form of minute dots not unlike flea-bites, slightly elevated above the surface, and having a tendency to coalesce in crescentic form. When the disease is very severe the eruption assumes a dark purple colour, and all the symptoms are exaggerated. *Defervescence* takes place on the *disappearance* of the eruption, but not on its *appearance*, as in small-pox. Desquamation occurs in the form of very fine branny scales.

During the course of the disease a troublesome cough annoys the patient, in fact this is often the most distressing symptom, but this rapidly disappears as the eruption fades; nothing is more constantly present, and, with the exception of the eruption, more characteristic. The temperature rarely exceeds 103° to 104° , and about the tenth day reaches the normal standard.

Bronchitis, pneumonia, diarrhoea, and in severe cases, gangrene attacking the mouth, vulva, nose and lungs, may

Catarrhal symptoms, with a harsh, clanging cough, as in measles, are very frequent. One of the most constant premonitory signs known is sore throat, and cases have occurred in which there was inflammation of the tonsils, velum, and uvula. The duration of the invasion stage varies from a few hours to three, four, or five days, and it is succeeded by the appearance of the characteristic rash. The eruption appears usually about the second day, many authorities say on the first, though it may not be seen until the fourth or fifth. It consists of an efflorescence breaking out in some cases at once all over the body; in others, beginning on the sides of the nose or face and then spreading quickly over the body, and consists of small red spots, which run together to form patches of variable size, with irregular margins, not of so distinctly crescentic a shape as those of measles. The eruption is usually at its height on the second day, rapidly disappearing in three or four days (*Bristowe*). In colour, the patches have been aptly compared by Paterson to the appearance "produced by a writing quill dipped in red ink and having its point placed on moist white paper." In some cases, the patches may so coalesce as closely to resemble the eruption of scarlatina, but careful examination will always discover one or more distinct patches. The eruption is attended with considerable itching. A rash not unlike that of *rötheln* is often caused when *copaiba* or *chloral hydrate* is taken for some time. While the eruption continues, the other symptoms increase, the sore throat is aggravated, and there may be much hoarseness and loss of voice, with considerable external swelling and tenderness. There is also occasionally much internal tumefaction, with total inability to swallow even the slightest portion of fluid, which generally regurgitates by the nose.

The pulse during this stage is frequent, the skin hot and dry, and the temperature rises to about 101°F. Death may occur during this stage by suffocation from the great mucous secretion in the throat. Vomiting is not uncommon.

The eruption stage lasts a variable time—from eight hours to eight or ten days, the average being about four or five days. On the rash disappearing, the third stage, that of desquamation, takes place. This commences at the centre of each eruptive patch, and extends towards the circumference. It is of a branny furfuraceous character, the epidermis never being shed in large scales or pieces, as in scarlatina. This process takes from five to twelve or fifteen days.

Diagnosis.—The only affections with which rōtheln can be confounded are measles and scarlatina. From them it differs, as will have been seen, in the general mildness of the symptoms; in not being accompanied by so high a temperature; in the appearance of the rash at once on the whole body and its characteristic appearance; in the manner of desquamation; in the size and brightness of the eruptive patches increasing with the severity of the attack; and in the tongue being more or less dirty at first, then strawberry-like, and finally smooth. Fagge was inclined to doubt the existence of rōtheln as a distinct disease, and suggested that it is possibly an anomalous or ill-developed form of measles, a view negatived by the facts that an attack of rōtheln affords no protection against either measles or scarlatina, and, *vice versa*, neither measles nor scarlatina protect against rōtheln.

Treatment.—In most cases little treatment is required, confinement to bed, an aperient, generally a simple saline mixture, being usually all that is necessary. The throat symptoms may require attention, and for their relief steaming with hot water and a simple antiseptic gargle may be

WHOOPIING-COUGH.

Lat. *Pertussis*. Fr. *Coqueluche*. Ger. *Keuchhusten*.

DEFINITION—An epidemic specific disease, occurring most frequently among children, and characterised by a peculiar whooping noise at the end of a fit of coughing, the whoop being produced during inspiration.

Pertussis—*pertussis*, a continual cough,—is a disease of early childhood, in rare cases occurring in adults. To children under five years of age it is very fatal. No anatomical appearances present themselves after death beyond those which attend ordinary bronchitis. The disease may be described as a cough having a more or less paroxysmal tendency, often assuming a suffocative character, and attended during the respiratory movement with the characteristic whoop. The disease is directly contagious from person to person, and occurs oftener as an epidemic than in isolated cases, one attack generally giving complete immunity from further visitations. The specific virus has not yet been discovered. It may also spread by infecting rooms, schools, &c. Whooping-cough frequently follows, but sometimes precedes an epidemic of measles. It also commonly affects children during the first and second dentitions. It sometimes attacks adults and may be very serious in the aged.

Symptoms.—The first stage of the disease, generally lasting ten or twelve days, in some cases is that of a common catarrh, accompanied with an obstinate and persistent cough; profuse expectoration of a clear viscid mucus succeeding each paroxysm. In others (more rare) the premonitory symptoms are those of bronchitis, attended with great fever and dyspnoea. These symptoms are

succeeded by the *convulsive stage*, which may last three, four, or more weeks, with its characteristic cough. The paroxysms become more convulsive and begin with a series of violent *expiratory* acts, which follow each other so quickly that inspiration is for a time prevented. The face becomes purple, and the eyes project and are filled with tears. Just as the little patient appears on the point of suffocation, a long and difficult inspiration—the harbinger of the patient's safety—now takes place; the air as it rushes in through the half-closed glottis giving rise to the peculiar crowing sound of whooping-cough. The skin during the fits is sometimes bathed in a cold sweat. An attack of vomiting, or the expulsion of some glairy mucus, generally terminates the paroxysm. The child having got rid of its last meal is ready to begin another. In fact, children often attacked during a meal vomit, and recovering from the paroxysm return to their meal as if nothing had happened. There may be four or five of these attacks in the day, or much more frequently in some cases. Hæmorrhage from the nose and an involuntary discharge of fæces frequently take place during the paroxysm. Ulceration under the tongue is common, and there may be temporary glycosuria and pain in the pectoral muscles after the fits of coughing. The respiratory murmur is absent during the fits. The most frequent complications which may occur during the course of this disease are those which relate to the respiratory organs on the one hand—pneumonia, bronchitis, interstitial emphysema, pleurisy, and broncho-pneumonia, often with collapse; and on the other, the brain and nervous system—congestion of the brain and convulsions. Though rarely fatal in itself except in very young children, it becomes so when complicated with any of the above-mentioned affections. The stage of decline is marked by a gradual diminution in the number of the paroxysms and a slow convalescence.

Diagnosis.—Whooping-cough must not be mistaken for acute bronchitis: this can only be done in the earlier stage of the disease, for the whoop is characteristic in the later stage. It must also be separated from laryngismus stridulus, and from tuberculosis of the bronchial glands at the bifurcation of the bronchi. Niemeyer says, "If a child has a violent, *prolonged* cough attended with *vomiting*, suspect and treat as if it were whooping-cough."

Prognosis.—It is a very fatal disease to poor and delicate infants. It is said to rank third among the fatal diseases of children, but in some towns it is even higher.

Treatment.—Nearly every drug in the Pharmacopœia has at some time or other been declared a specific. As the curative measures appear so unproductive of good results, we may try what can be done by way of prophylaxis. Taking into consideration the epidemic character of the disease, children should, if possible, be removed from the infected locality, especially if they are delicate or strumous. In the early stages of the disease confinement to one room, in which a uniform temperature is maintained during day and night, should be enjoined, and all exposure to cold avoided, although plenty of fresh air should be arranged for. A trip to the sea-side will often effect a cure when all other means have failed. Vaporising various volatile antiseptics, and especially a solution containing creosote and carbolic acid, should be carried out continuously in the sickroom. If drugs be employed, belladonna, together with bromide of ammonium or small doses of quinine, and the cautious administration of antipyrin will be found to afford the greatest relief. There is one remedy which is invaluable in the treatment of this disease, and that is hydrocyanic acid. We frequently combine it with the tincture of belladonna and ipecacuanha. Recently $\frac{1}{2}$ to 1 minim of bromoform dissolved in oil or mixed with spirit and water has been

specially recommended for relieving the spasm. Friction down the spine, with a mixture of the compound camphor and belladonna liniments, is also useful. A substitute for Roche's embrocation may be used, and is given among the prescriptions at the end of the book. During the third stage, that is, when the disease begins to decline, a generous diet, with eggs and wine, accompanied with the exhibition of ferruginous tonics, is indicated.

PAROTITIS.

Lat. *Parotides*. Fr. *Oreillon*. Ger. *Ziegenpeter*.

DEFINITION—An epidemic and contagious affection of the parotid gland.

Parotitis, or Mumps, is an inflammatory affection of the parotid gland, probably induced by a specific poison, the nature of which is unknown. The period of incubation is from two to three weeks. Enlargement of the gland or glands, for both sides are often affected, the one following a day or two after the other, takes place, accompanied with a sensation of tenderness when touched and pain during mastication. The submaxillary glands may also be involved. The pain shoots up towards the ear, the lower part of which is pushed outward by the enlargement of the gland. The attack is generally accompanied with catarrhal symptoms, slight fever, and pain in the head, together with loss of appetite, foul breath, furred tongue and general malaise. Suppuration seldom takes place, and the duration of the attack is short, with no tendency to become chronic. Towards the end of the glandular affection the testicles in males and the ovaries or breasts in females sometimes become tender and swollen, and you may leave a patient on one day almost well, to be summoned on the next to an enlarged and painful testicle.

The disease is contagious, and occurs usually but once during life. It is most common in spring and autumn, and chiefly attacks children and adolescents.

The Prognosis is favourable, though permanent deafness may result in one or both ears, probably from pressure of the swelling on the auditory nerve.

Treatment.—In most cases the administration of an active purgative in the first instance, followed where necessary by a few minims of tincture of aconite repeated every two or three hours till the fever abates, are sufficient. The application to the gland either of warm anodyne fomentations or the icebag are of service; certainly heat is more kindly to the patient, and a hot bath is peculiarly soothing when metastasis threatens.

SYPHILIS.

Lat. *Syphilis*. Fr. *Syphilis*. Ger. *Syphilis*.

Pathology, &c.—Syphilis is the type of a truly contagious constitutional disease, *direct contact* with the virus being necessary for its origin and spread, infection through the air (as in some other specific fevers) being fortunately impossible. It is propagated by direct contact with a primary sore, by kissing, by nursing, by skin grafting, by vaccination if human vaccine be used, by using eating and drinking utensils which have been contaminated by a syphilitic person, and it has even been maintained that the semen of an infected man injected into the vagina of a healthy woman, irrespective of pregnancy, may propagate the disease. The virus exists in the primary sore in all the secondary lesions, moist papules, mucous patches, &c., and in the blood during certain periods, but is absent in those of the tertiary stage.

Any part of the body may suffer if the conditions for infection are favourable. Physicians, mid-wives, &c., may be infected on the finger, &c., through an abrasion, or even through an injured hair-follicle. The normal secretions—mucus, saliva, &c.—are not infective unless they are mixed with pus or blood. Not only is syphilis propagated by direct contact, but it may also be inherited, both the spermatozoa and the ova being important carriers of the contagion. It is a specific fever accompanied with local eruptions of varying character, and differing from other specific fevers mainly in its slower evolution, and in the more prolonged duration of its various stages.

A thin spirillum called the *spironæma pallidum** is present in large numbers in all the primary and secondary lesions of the acquired disease and, in still greater numbers, within the liver, spleen and other organs in the hereditary form. Its artificial cultivation has so far failed, but inoculation of the virus from a syphilitic lesion in man reproduces the disease in apes, their susceptibility rising in proportion as their conformation approaches that of man. The evidence so far available is highly presumptive, though not yet conclusive, that this spirillum is the causal germ of syphilis. There are several stages in the acquired form of the disease, known under the terms primary, secondary, and tertiary; the *first*, the primary or initial lesions, when the disease is limited to the part inoculated and the lymphatic glands connected with it; *secondary*, the secondary lesions, when after one to three months it affects parts not directly inoculated; and *tertiary*, the tertiary lesions, when after an interval of months or years of apparent health the disease returns with one or more of its recognised manifestations. Constitutional syphilis of the child, derived during foetal life from one of the parents, is known as hereditary syphilis. When the signs are manifest

* It is actively motile, from 4 to 14 μ long and about $\cdot 25 \mu$ thick, showing usually six to eight small, sharp, regular curves, and is tapering and pointed at both ends. It was first described in 1905 by Schaudinn and Hoffmann under the name of *spirochæta pallida*.

at the time of birth it is spoken of as congenital, but the more general term "hereditary" is the better.

THE PRIMARY LESIONS.—The primary indurated area or papule (chancre) which appears at the seat of inoculation is due to a proliferation of the connective tissue cells in the immediate neighbourhood of the smallest blood-vessels in the papillary layer of the cutis vera. There is also an emigration of leucocytes, a thickening of the media, and a marked proliferation of the intima, which rapidly goes on to obliteration of the lumen (an acute obliterative endarteritis). The proliferation and infiltration continue until all the cutis and sometimes even the subcutaneous tissue is equally affected. The covering epithelium gets thinned and disappears (erosion) and the indurated areas break down (ulceration). Imperfect fibrillation, epithelioid and sometimes giant cells are to be seen in old chancres. Similar hyperplastic changes to those in the cutis appear in the adjacent lymphatic glands.

THE SECONDARY LESIONS.—Symmetrical small superficial ulcers on the tonsils, symmetrical eruptions on the skin, mucous patches, affections of the eye, &c. They closely resemble other inflammatory processes, and are due to a wide-spread distribution of the syphilitic contagion throughout the body. This contagion is at first confined to the site of inoculation, from which it spreads by the lymphatics only to the nearest lymphatic glands, thus causing the primary lesions. It then escapes into the circulation and thus reaches distant tissues, causing the irritative phenomena of the secondary lesions. Its infective power gradually diminishes, so that, speaking generally, it is greatest in the beginning (the primary chancre) and least in the late secondary manifestations, entirely losing it (usually) by the time the next, or tertiary, stage is reached. While it is generally true that it has lost the power of propagating the disease in a healthy person after two years (the period usually imposed upon a syphilitic who contemplates marriage), yet rare cases of infection are known after much longer periods.

THE TERTIARY LESIONS.—Circumscribed tumors, known as gummata, diffuse indurations of the viscera and nervous system, inflammatory changes in the blood-vessels, and waxy degeneration. The histological changes are similar to those which occur in the chancre. They are best studied in the gumma, which is the most characteristic lesion of this stage. It varies in size from a pin head to a walnut or bigger. It is generally found around a blood-vessel, which shows marked endarteritis. In its early stage it consists of proliferated connective tissue cells and leucocytes. Later, some of the cells get larger (epithelioid), and one or more giant cells may appear. The margin generally shows marked fibrosis. Degenerative changes, fatty or granular, subsequently appear, and the gumma may become largely or entirely absorbed.

Symptoms.—After a period of incubation, lasting on the average from about three weeks to a month, a slight hard and indurated sore appears, generally under the foreskin, but sometimes on the body of the penis. After from a few days to a week or two the lymphatic glands of the groin become enlarged, accompanied with more or less disturbance of the general health, which, however, is generally very good in this stage. The enlarged glands are not painful, and seldom suppurate when the infection has been pure. The primary sore or chancre is a minute papule or vesicle of a dusky-red colour, with an *indurated base*, unattended with pain or itching. After a time a thin greyish crust forms, which may be shed several times till at last the sore has an elevated dusky-red margin surrounding a concave excavation with a grey dry surface. The ulcer exudes a scanty secretion. The sore finally disappears, leaving a cicatrix behind. But in some cases there is a very small and sometimes even no primary sore to attract attention, and consequently no cicatrix. The secondary symptoms, "*stage of eruption*," generally

appear after about six weeks, although they may not appear for three months, rarely later, after the healing of the primary sore, and are then ushered in by well-marked febrile disturbance, and often with neuralgic pains in the head—"bitemporal neuralgia"—and in other parts of the body, becoming more severe as night approaches. The fever is usually mild, not rising above 101°F . It is sometimes remittent in type, and sometimes intermittent, and occasionally severe, even up to 105°F . A macular eruption or roseola, or a papular, scaly, pustular, &c., may now appear on the body in successive crops on and off for two or three months. One marked peculiarity of syphilitic eruptions of this stage is their "*polymorphous*" character—thus we may have scaly, papular or tubercular, vesicular or pustular eruptions more or less congregated together. Other specific characters of these eruptions are their frequently coppery colour and their aptitude to affect those parts usually avoided by the non-specific eruptions they resemble, and also their tendency to arrange themselves in circles or semi-circles. Simultaneously ulcers form on the tonsils, which are usually symmetrical and give but little pain. They do not tend to spread much, and usually heal after a few weeks. Mucous patches, areas of inflamed mucous membrane, may be seen at angles of the mouth, on pharynx, palate, &c.; when considerably raised above the surface they are called condylomata. They are seen in the mouth, pharynx, around anus, &c. The hair may fall off, either in patches or by a general thinning, and transient alopecia result. In some cases the eyes, usually one before the other, are affected with iritis, attended with the formation of yellowish-red nodules of lymph. A zone of ciliary congestion is usually well-marked, though it is slight in mild cases. In severe cases there is great pain. A retinitis of a very insidious nature may follow. The bones, especially those of the skull; may also become affected, and the most painful of these affections are often to all

appearance merely slight periosteal swellings. The pain is most severe at night, and, according to Bäumler, is due to the determination of blood to the affected parts. This determination of blood to the local periostitis accompanies the febrile rise in temperature which frequently takes place at night. A pronounced anæmia is also frequently seen and the patient may lose flesh. The symptoms of the secondary stage rarely last after eighteen months have passed.

Gliding almost imperceptibly from the second stage, and carrying with it many of the affections of that stage, the *tertiary* commences. The general health becomes more and more deteriorated, and severe complications now arise, there being a marked tendency to the formation of tumors in the several organs of the body. These tumors are especially met with in the early stages of congenital syphilis, and in the later stages of the acquired form. The tumors, on account of their gummy, semi-fluid contents, are known as "*gummy tumors*" or, shortly, "*gummata*." The internal organs most frequently attacked by them are the liver, testicles, skin and subcutaneous tissues, and brain membranes. They also appear in most of the other viscera and the muscles, in which they are, however, comparatively rare. They have a reddish-white, translucent, gelatinous appearance; but in their later stages they become firmer, yellowish, and cheesy-looking, and on section have an appearance similar to the section of a horse-chestnut. The symptoms to which these tumors give rise will depend on the organ in which they are located. In the skin and subcutaneous tissue they are apt to break down and form deep ulcerations. These tend to be serpiginous, healing at the centre and spreading at the margins. They leave characteristic cicatrices, often coppery in colour. Similarly, deep-spreading ulcerations occur upon the mucous membranes—*e.g.*, pharynx, larynx, œsophagus, rectum, &c. The ulcerations may be very destructive and the subsequent cicatrization extensive, causing great deformity or stricture, as in gullet

and rectum. In the solid organs gummata form encapsulated masses, or fibroid areas, or cicatrices, resulting in various puckering and deformities. Another lesion characteristic of tertiary syphilis is a form of interstitial inflammation. This is seen in the liver, lungs, bones, and nervous system. It may be diffuse, or more or less localised. Changes in the arteries are also met with. An endarteritis of the smaller arteries and veins, resulting from a proliferation of the cellular constituents of the intima, is the most frequent. The newly formed tissue bulges into the lumen, which it may fill up completely, or obliterate by causing thrombosis therein. The media and adventitia are also affected and show cell infiltration. A periarteritis nodosa or gummatous formation is sometimes also seen, especially in the cerebral arteries. Aneurism also may be due to syphilis.

Waxy degeneration is not infrequent in acquired but rare in hereditary syphilis. Inflammation of the periosteum and bone are very common in tertiary syphilis. They specially affect the more superficial bones, and may be acute and result in suppuration and necrosis, or be chronic and cause much thickening. Certain lesions, not perhaps exclusively caused by syphilis, should also be mentioned, viz., locomotor ataxia, general paralysis, certain types of epilepsy, and arteriosclerosis.

The eruptions on the skin accompanying constitutional syphilis, as before said, present certain peculiarities. They have a tendency to become circular or semi-circular, seldom angular. Those belonging to the *secondary stage* have a *bilateral tendency*, are superficial, and do not, as a rule, leave a scar; they are often polymorphous—that is, we may have pustules, papules and vesicles side by side, and these may assume a coppery hue. In most cases the eruptions do not itch.

The eruptions of the *tertiary stage* are non-symmetrical, attack the deeper structures, which they destroy, and leave permanent scars.

Hereditary Syphilis.—This may be derived from father or mother, or from both. Parental syphilis is an important factor in the production of repeated abortions. Should the child be born alive it is often puny; as it grows its face assumes an ancient or "old man" look, it is pale and anæmic, and the skin has not the healthy baby hue. In some cases the child is born with an apparent nasal catarrh, or the "snuffles," and it may suffer from certain skin eruptions on the buttocks, ankles, wrists, or hands. There may be ulcers on the lips, fissures at the angles of the mouth, and the liver and spleen enlarged. In other cases the child is born to all appearance healthy, and it is not till some weeks—from the fourth to the eighth, rarely later—after its birth that it shows any signs of its syphilitic inheritance. Snuffles, skin eruptions, lip fissures, mucous patches, &c., appear. The reactions from these lesions are very contagious and may infect the wet nurse if she be other than the mother of the child. The mother who has given birth to a syphilitic child cannot be infected by it (Collis's law). During the second dentition, the central upper incisors become peculiarly notched and peg-shaped. They are narrower at the cutting edge—which shows a single, usually shallow, notch—than at the root, as first pointed out by Mr J. Hutchinson. General growth and development is slow. The forehead is prominent, the bridge of the nose is flattened, and puckered lines run from the corners of the mouth. Keratitis, iritis, bone, and other lesions are met with.

Diagnosis.—The general diagnosis of syphilis will rarely be difficult. The patient, either from ignorance or design, may give unsatisfactory answers, and careful enquiry should be directed to the existence at any time of sore throat, skin rashes, falling out of hair, as well as the primary sores. The mouth, throat, and skin must be carefully scrutinised for signs of syphilitic lesions. The bones should be felt for

thickenings. Frequent miscarriages are suspicious. The effect of anti-syphilitic treatment is of much value.

Prognosis.—The primary and secondary lesions readily heal, especially under appropriate treatment. The tertiary lesions may be rendered very mild by appropriate early treatment, and even when severe they generally yield to proper treatment; but certain visceral, nervous, and other lesions, which cannot be entered into more fully here, may run a serious course and end fatally.

Treatment.—Perhaps in no other disease must the personal equation of the patient be so closely studied. Many patients appear to be much more immune, or at least resistant, to the manifestations of syphilis than others, but a mild case to begin with may develop the most severe and disastrous tertiary phenomena. Treatment, therefore, should and must continue for at least two years and probably long after the manifestations of the disease have apparently disappeared for the last time.

The primary lesion is not now, as a rule, either excised or cauterised, because the infection is known to be much too rapidly disseminated throughout the body. Cleanliness is all that is held to be requisite in most cases.

There is much difference of opinion as to (1) whether it is better to begin treatment at once or to wait for rashes or other manifestations to appear, and (2) whether the treatment when begun should be continuous or should be interrupted by periods when no medicine need be taken.

Mercury is the remedy for secondary syphilis, and it may be administered by mouth, by inunction, by vaporisation, or, lastly, by hypodermic injection. Grey powder in 2 to 5 grain doses, blue pill or perchloride of mercury in the form of the liquor ($3j = \frac{1}{16}$ grain being a suitable dose) are the usual forms of mercury administered by mouth,

and they are sometimes combined with a small dose of opium. Mercury may be administered night and morning, and, in any case, care should be taken to avoid disturbing digestion in dyspeptic subjects.

For inunction, about a drachm of unguentum hydrargyrum may be rubbed into the skin of the trunk, thorax or inner aspects of arms or thighs every night at bedtime, and kept up for a week, when a hot bath should take the place of the inunction every sixth or seventh night. A different area of skin should be selected each night, and if the inunction is carried out by a professional rubber, the hands must be guarded to prevent absorption. Any other mercurial ointment may be substituted, perhaps the red iodide being the one we should prefer.

Vaporisation is not so efficacious, but it seems to suit some cases better than inunction, or it may for a time be substituted for it. It consists in the vaporising of 20 to 40 grains of calomel along with a steam bath. A chair, a waterproof sheet with an arrangement by which the patient's head is left free, and a steam kettle and lamp with a small capsule, upon which the calomel is placed, are all the requirements. Hypodermic or rather intramuscular and sometimes intravenous injections have been commended, but they are most undesirable, and except in the most urgent cases should never be resorted to. Where considered necessary $\frac{1}{10}$ to $\frac{1}{8}$ grain of perchloride of mercury may be injected in solution with water.

When mercury is being administered always examine the teeth and gums regularly, inculcate absolute cleanliness and daily use of the tooth brush, and give an alum mouth wash. Should salivation begin, stop the treatment until it has disappeared. There are individuals whose idiosyncrasy prevents the administration of mercury, just as there are persons who cannot take iodide of potash.

In the tertiary and even late secondary stages of syphilis, potassium iodide finds its great sphere of usefulness. It

should be given in 10 to 20 grain doses thrice daily, and may be persisted in till iodism appears. Sometimes increasing the dose arrests iodism, but very often an occasional interval of a few days or a week or two prevents its appearance at all. In many cases mercury and iodide may be taken combined. A sea voyage to last some months is of very great value and improves the general health, which, as a rule, suffers very severely, and marriage should be absolutely forbidden until the lapse of two years at least. For hereditary cases mercurial inunction and small doses of grey powder are specially applicable. An infant's binder may be saturated with a mild mercurial ointment. A pregnant woman who is syphilitic should be vigorously treated upon the lines already laid down.

Lastly, never forget that a syphilitic patient is always below par and very generally anæmic, and successful treatment includes the use of tonics, and especially iron, in addition to the specific remedies referred to above.

2.—ENDEMIC.

TYPHUS FEVER.

Lat. *Typhus*. Fr. *Typhus*. Ger. *Exanthematischer Typhus*.

JAIL FEVER, SHIP FEVER, CAMP FEVER, HOSPITAL FEVER,
OCHLOTIC FEVER (οχλος "A CROWD"), &c.

DEFINITION—A continued fever, characterised by sudden accession, great prostration, and a general dusky, mottled rash, without specific lesion of the bowels.

Typhus fever has been long known, having been described by Fracastorius and Cordanus, but it has only in comparatively recent times been distinguished from typhoid fever, with which it was confounded. It is a contagious continued fever, having a duration of from fourteen to

twenty-one days, attended with great depression of the vital functions. The tendency to cerebral complication is great, hence this disease has been called by some *brain fever*.

Typhus is also characterised by the appearance, on or about the fourth or fifth day of the fever, of a peculiar maculated eruption on the skin.

Etiology.—(1) PREDISPOSING. (2) EXCITING.

Predisposing Causes.—Poverty, overcrowding, with imperfect ventilation and unwholesome food, together with mental or bodily exhaustion, form the chief predisposing causes. Sex does not predispose to typhus, but with regard to age typhus is, as a rule, a disease of adult age, especially after thirty. Some authorities say that children are as frequently attacked as adults. It is also a disease of the poor. The seasons do not appear to influence its spread, epidemics occurring at different periods of the year. The temperature and hygrometric state of the atmosphere influence it but little. It may be said that, as a general rule, any cause which lowers the vitality of the individual, added to overcrowding—*ochlesis*—favours the origin and spread of typhus.

Exciting Cause.—The exciting cause is a specific poison. Several microbes have been described as occurring in the blood and tissues, but the specific organism has not yet been discovered. The poison may be retained by fomites for a long time. It is highly contagious, often attacking doctors and nurses in attendance upon the sick.

Geographical Distribution.—Typhus is found in all European countries, in North America, and probably in India. It does not appear to have been found in Australia, New Zealand or Africa. It is most common in the British

Isles. It has been the scourge of armies, the French army suffering severely in the Crimea. The Bavarian army in Bohemia during the Thirty Years' War lost 20,000 men. The fatal mortality of the six "Black Assizes," the last in 1750, was due to typhus.

Morbid Anatomy.—There is no very characteristic change. Peyer's patches are not ulcerated. The blood is dark. The liver is soft, and on section shows a dull clay-like lustre.

Symptoms.—The invasion of this fever is in most cases *abrupt*; sometimes, however, gradual and obscure, one or two days of slight indisposition ushering in the attack. As a rule, however, fits of shivering, attended with a severe headache, pains in the back and limbs, mark the onset of an attack. During the *first week*, symptoms common to most acute diseases make their appearance. There is loss of appetite, the face is flushed and dusky, the eyes injected and watery, tongue furred, then dry and feverish, the skin hot; there is great thirst, a great sense of chilliness, a full, quick, compressible pulse, 100 to 120; constipation, scanty high-coloured urine, and a sensation of intense lassitude and disinclination to move about. The demand for cold water is urgent. The temperature rapidly rises, 104° to 105° , the maximum being reached between the fourth and seventh days. A high range of temperature during this week—generally presages severe cerebral symptoms during the second week. The respiration is sometimes impeded and accompanied with frequent sighing, but, as a rule, it is increased, the average being about 21 per minute. The muscular debility is usually so intense that the patient takes to his bed on the second or third day of the fever, and in most cases is incapable of moving from it without assistance. The expression on the face soon becomes dull, heavy, and stupid. A peculiar roseolar rash, which may be mistaken for that of scarlet fever, in some rare cases precedes the

characteristic rash of typhus. From the fourth to the seventh day, usually on the fourth, the characteristic rash of typhus makes its appearance. The *maculæ*, *mulberry* or *rubeoloid rash*, occurs under two forms—as distinct spots or as a subcuticular mottling. The spots first appear of a dusky pinkish-red colour, slightly raised above the skin, and disappear on pressure; but after the first and second day they become dark and more dingy, less elevated, and do not disappear on pressure, only becoming slightly paler. On the eighth or tenth day true petechiæ are in some cases formed. Petechiæ may be defined as minute purplish spots or subcutaneous ecchymoses, which do not disappear on pressure. Flea bites have been mistaken for the petechiæ of typhus; the former are more circumscribed, disappear on pressure, and have a central puncture which does not disappear when pressed. The rash is most abundant in severe cases, and never appears in successive crops as in typhoid fever. Within forty-eight hours from its first appearance the rash is fully out and varies in colour on different parts of the body, giving a mottled aspect to the skin. It may be said to go through three stages:—

1. Pale dirty pink or florid, slightly elevated, and *disappearing* on pressure.
2. Reddish-brown or rusty, not elevated, and *slightly disappearing* on pressure.
3. Livid and petechial, *not affected* by pressure.

It first appears on the front of the axilla and sides of the abdomen, then on other parts of the body, and then subsides between the fourteenth and twenty-first days of the fever. In adults the rash is seldom absent, but in children its absence is more frequent. During the *second week* the symptoms of the first week become more aggravated, the intellect becomes affected, and there is much confusion of ideas. About the seventh day, low muttering delirium

occurs, "typhomania," or the patient may become excited and noisy, making frequent attempts to jump out of bed. Niemeyer mentions a case in which a patient suffering from typhus was sent to him from jail, in a strait-jacket. During the early stages of the delirium, which may last two or three days, the patient may be restored to consciousness by a sharp question; but as the fever progresses he becomes less easily roused, more stupid, and his countenance wears a half-drunken expression, at no time betraying anxiety. The nervous excitement increases towards night, and the prostration towards morning. The surface of the body, particularly the face, assumes a dusky or dingy hue. As the disease progresses the tongue becomes covered with a thick brown or black coating, sordes appear on the teeth, and the lips are dry and cracked. He is now unable to move himself, lies perfectly prostrate in bed, entirely indifferent to everything going on around him, and his motions and urine are passed involuntarily. At other times he is found unconsciously picking or fumbling with the bedclothes. Subsultus tendinum is also frequent. Diarrhoea if present is slight, and there is little if any abdominal tenderness, but sometimes slight tympanitis. There is usually more or less catarrh, attended with slight cough, proceeding from subacute bronchitis. The pulse varies little from day to day, is feeble and quick, reaching 110 and 120 beats, and is rarely dicrotinous as in typhoid. The temperature is, as a rule, lower than during the first week, being generally a degree or two higher in the evening than in the morning. The maximum temperature is usually reached by the fifth day. Permanently high morning and evening temperatures alone betoken gravity, and a rapid rise to about 108°F. usually precedes death. The breathing during the second week is, as a rule, hurried, being from 30 to 40 per minute. The urine is generally high coloured, the urea and uric acid being increased, while there is a gradual diminution of the

chlorides, which may not exceed two or three grains in twenty-four hours, and in some cases may even be absent. Albuminuria is also not infrequent, but nephritis is rare. In the advanced stages of typhus, even during active delirium, the pupils are *contracted* and insensible to light (Murchison). During the stage of coma they may, however, become dilated. This condition of the pupils distinguishes typhus from typhoid fever. About the fourteenth day sudden amendment in the condition of the patient may take place, a refreshing sleep stealing over him, from which he awakes conscious, but fearfully weak. A rapid decline of temperature betokens the setting in of convalescence. In fatal cases death takes place about the twelfth or fourteenth day, the coma becoming more profound as death approaches. The odour from the body is said by many authorities to be distinctive and often most offensive. Severe bedsores may form on the sacrum and hips; and the extremities—the nose, ears, &c.—may become gangrenous. Pregnant women if attacked do not, as a rule, abort, and if the child is born alive it does well.

Several complications may occur during the course of the fever; these are generally to be referred to the cutaneous, nervous, respiratory, and circulatory systems:—

Cutaneous System.—Among the true exanthematous fevers, the absence of a rash, as a rule, betokens a mild attack. The opposite is, however, the rule with typhus. In the exanthematous fevers the eruption soon disappears; in typhus it is persistent.

Nervous System.—Convulsions, due to the blood-poisoning, are fortunately of rare occurrence. Increased cerebral excitement, ending in delirium more or less severe, generally supervenes towards the end of the first week; in non-fatal cases, the delirium passes off in three or four days.

Respiratory System.—Pneumonia, generally in the lobular form, is of frequent occurrence, sometimes ending in gangrene of the pulmonary tissue. Bronchitis is

common, and the patient is frequently troubled by a dry cough with slight expectoration.

Circulatory System.—Dr Stokes has described a peculiar form of cardiac lesion, which he has named “typhus softening of the heart.” Sometimes the muscles of the heart are found to have undergone the hyaline change described by Zenker as frequent in typhoid. It is merely a form of coagulative necrosis. A diminution of the impulse and an impairment or loss of the first sound indicate the cardiac phenomena of typhus.

The Prognosis must be formed on a careful review of the symptoms, the age and condition of the patient being taken into consideration. Death generally occurs from a combination of asthenia and coma. The mortality is from 12 to 20 per cent., although it sometimes reaches 50 per cent. in older people.

Diagnosis.—Often very difficult in sporadic cases. It is frequently mistaken for typhoid, for severe cerebro-spinal meningitis in its early stage, for measles, &c.

Treatment.—The treatment has now to be considered. Sydenham’s remarks on the treatment of fever are worth recording here:—“I cannot bring my brain to comprehend the meaning of those physicians who are continually talking about the administration of remedies that promote the concoction of the febrile matter, points which they insist upon when called in at the beginning of the diseases. At the same time, they have no hesitation in recommending medicines that are intended to control the fever. Why! the fever itself is nature’s instrument by which she separates the pure parts from the tainted ones!” He himself recommends the treatment now adopted, and best described as the “expectant treatment.” The patient should be put to bed in a well-ventilated room at the top of the house.

It is difficult to get too much ventilation for typhus patients. Free ventilation, as a rule, prevents the spread of the disease, and also, to a great extent, secures the safety of the attendants. The utmost care should be taken of the patient's food—milk, beef-tea, custards, and so forth should be administered at regular intervals (2–3 hours); and the nurses should be instructed to note carefully the hours when food is taken, the condition of the patient's pulse and respirations, and lastly the number and hours of evacuations of the bowels. A water-bed should be used, and the skin of the sacrum kept absolutely clean and dry so as to guard against the formation of bedsores. Washing the skin with spirit and water or a solution of alum once or twice a week is very serviceable as a preventive measure. Bedsores are best treated with charcoal poultices or boric acid lotion. The bladder should be frequently examined for retention of urine, so common in this disease. The fact that the patient passes a considerable quantity of water unconsciously should not prevent a frequent examination of the bladder, which may be only overflowing. In doubtful cases pass a soft rubber catheter. A mixture consisting of dilute hydrochloric acid, $\text{m}15\text{--}20$, with syrup of lemons and water, is found most useful in relieving the parched tongue, mouth and throat, thus increasing the salivary secretion, and supplying to the gastric juice an element which is frequently deficient in fever. Medicinal antipyretics should here be used with caution, owing to the frequent tendency in typhus fever to cardiac weakness. Beware of doing too much. Cooling drinks to allay the thirst, and tepid sponging of the body, together with hygienic and supporting measures, will form the most important part of the treatment of typhus. Be careful also in the use of alcoholic stimulants; do not, because it is a case of fever, rush to the brandy bottle; and remember that the occurrence of delirium does not always prohibit the use of alcohol, the delirium often being due to extreme prostration. When shall we use stimulants? As

a rule, wait till the second week of the fever. But if at any time before or after that period there is marked exhaustion, the first sound of the heart becoming obscure, with a feeble pulse, stimulants should be given, strychnin hypodermically, ether, or alcohol beginning with two tablespoonfuls of brandy or whisky and carefully watching its effect. Continue the alcohol if you find "that it agrees with the patient." Dr Haughton maintains that "the effects of alcohol administered in fever, when the temperature does not exceed 105°F. , are twofold—immediate and secondary. The immediate effect is to supply a hydrocarbon to the blood, which is decomposed by it in preference to the body tissues. The secondary effect of alcohol is to change the blood itself, which thus loses its oxidising qualities, in consequence of which the temperature falls, the hyperdicrotic character of the pulse disappears, and the distinctive metamorphosis of the tissues becomes lessened." Increased febrile movement and restlessness, with delirium, suggest the discontinuance of the alcohol. Hydrobromate of hyoscin ($\frac{1}{100}$ grain doses) is useful in checking delirium. Stokes of Dublin strongly recommends cold affusions, both to head and body, while an icebag kept over the head is of undoubted value. Constipation may be relieved by any simple aperient, and diarrhoea when it occurs can generally be kept in check by modifying the diet, such as boiling the milk, and restricting the patient to boiled milk with lime water. Bronchitis and respiratory complications should be treated by appropriate remedies. Tonics and change of air will be required during the period of convalescence, which is generally rapid. The best measures to prevent the spread of typhus consist in free ventilation, thorough cleanliness, and lessening the overcrowding which is an important factor in the production and spread of typhus. The patient should be isolated from the public for a month after the symptoms of typhus have disappeared, and during this time might with advantage take weekly two or three warm baths

containing one or other of the various suitable antiseptics, and the clothes of the patient ought to be thoroughly disinfected by means of dry heat, or destroyed.

YELLOW FEVER.

Lat. *Febris flava*. Fr. *Fièvre jaune*. Ger. *Gelbes fieber*. Syn. *Gelfieber*.

TYPHUS ICTERODE, VOMITO NEGRO, BULAM FEVER.

DEFINITION — A malignant epidemic fever, usually continued, but sometimes assuming a paroxysmal type, characterised by yellowness of the skin, and accompanied in the severest cases with hæmorrhage from the stomach (black vomit), nares, and mouth.

Yellow fever is essentially a disease of tropical climates, seldom extending beyond 40° north or 20° south latitude.

Etiology.—It is a disease of the sea coast, of cities, and of parts of cities, particularly the badly drained and crowded parts, being often singularly localised like cholera. A continued high temperature (72° to 77°F.) of some weeks' duration greatly influences its origin and spread. The negro race is much less susceptible but is not immune. The white races, and especially those recently entering the yellow fever zone, are most susceptible. It is stated by La Roche on the authority of Deveze that curriers, tanners, soap-boilers, candle-makers, and in general all those who habitually breathe an unwholesome atmosphere, are not liable to the disease. Dr Rush noticed a like immunity among butchers. On the other hand, locksmiths, bakers, and all those who habitually go near furnaces, are readily attacked by the disease (*Deveze*). Whatever may be the nature of the yellow fever poison, it has been clearly proved to be portable; but "the how" is another debateable

question. It is asserted that it can be introduced into a town by patients affected with the disease, or by *fomites*; but both these methods of spread are very doubtful. Improper food, drunkenness, sexual excesses, and neglect of proper hygienic measures are, as in other infectious diseases, conducive to its origin and spread. The period of incubation varies from two to seven days. It occasionally spreads and prevails epidemically in other regions, as in the Southern States of America, &c. Dr Guiseppe Sanarelli has described a bacillus which has the form of a motile rod with rounded extremities, often occurring in pairs, and which is known as the bacillus icteroides. He believes it to be the cause of yellow fever. A small fine bacillus has also been described by others. It is thought to enter the human body by mosquito bites and is found in great numbers in the intestinal contents.

Pathology.—The *post-mortem* examination reveals no special anatomical characters. The liver appears to be most frequently affected. The colour is unnaturally yellow, in some cases resembling phosphorus poisoning, in others fatty degeneration is more or less marked. On the mucosa of the œsophagus and stomach may sometimes be found ecchymoses and erosions, and the stomach usually shows acute gastritis. The kidneys show intense glomerulo nephritis and fatty degeneration. There is an excess of urea in the blood.

Symptoms.—A sensation of chilliness, with or without rigors, followed by supra-orbital headache, pains in the limbs and back, anorexia, constipation, vomiting of a limpid opalescent matter, together form the symptoms met with in an early stage of yellow fever. Should the disease continue unchecked, the vomit in a considerable number of cases assumes the characteristic black colour, and a fatal termination is to be apprehended. Black vomit is not necessarily

a fatal symptom, but it is present only in severe cases. The attack, however, in most cases comes on suddenly, without any premonitory symptoms. The vomited matter consists of blood changed by the action of the gastric fluids, and varies in colour from a claret or dark-brown to almost black, with a sediment like coffee grounds. The temperature of the body for the first few days rises to about 104° – 105° , or even 110° F. (*La Roche*). About the fourth or fifth day defervescence sets in, the thermometer marking a steady fall to, or below, normal. The pulse seldom exceeds 100, sometimes sinking as low as 40 or 30 beats a minute. Respiration is quick and superficial. The tongue, at first moist and more or less coated, becomes smooth, dry, and red like raw meat as the disease progresses. The urine is always acid in the first stage; during convalescence it is alkaline. Sometimes this secretion is entirely suppressed. The colour for the first few days of the fever is normal, but it soon assumes a sulphur or a primrose tint; it then deepens to yellow or orange, and should the patient recover, may appear dark brown or black. Albuminuria is of frequent occurrence. The skin in the course of the disorder assumes a yellow colour, whence the name. This jaundice is of hæmatogenous origin and is present in almost all the fatal cases, and in a limited number of the cases which recover. Occasionally coma and convulsions may occur, most probably due to uræmia. Delirium is sometimes present. Several forms of this disease have been described, such as:—

1. The algid form.
2. The sthenic form.
3. The hæmorrhagic form.
4. The purpuric form.

Diagnosis and Prognosis.—Yellow fever is distinguished from malarial fever by the absence of any affection of the spleen in the former disease, and also by the efficacy of

quinine in the latter, and by the examination of the blood; and from small-pox by the presence of the peculiar eruption of that disease. From the relapsing fever the diagnosis is made by examining the blood. Before the relation to the spirillum was known, a severe form of relapsing fever with jaundice that once occurred in Dublin was unrecognised, and described as "British yellow fever." The prognosis in a case of yellow fever should always be very guarded; the presence of black vomit is of evil omen. The absence or gradual disappearance of albuminuria is a favourable sign.

Treatment.—With regard to the treatment to be adopted, most observers agree that in mild cases the less interference the better. Mild purgation should be at once resorted to, and a hot mustard and water foot bath is of undoubted value. Dr James W. Martin recommends that in the first stages calomel and quinine should be given combined with capsicum. When hæmorrhage sets in, ice and tincture of the perchloride of iron in large doses may be tried, and bismuth, opium, acetate of lead and ergot are also recommended, but it must be kept in mind that all medicines are very unreliable in checking this form of hæmorrhage. Stimulants are indicated and should be freely given during the second stage when the heart's action shows any tendency to weakness or collapse. The gastric irritation must be treated on general principles—creasote in drop doses, small doses of dilute hydrocyanic acid, or ice to allay the sickness. Mustard plasters or small blisters applied over the pit of the stomach will often afford relief. Should the vomiting become incessant, it is best to give nutritive enemata until the gastric irritation is allayed, and in this way avoid irritating the stomach. Sternberg has given bicarbonate of soda, grs. 150, perchloride of mercury, gr. $\frac{1}{3}$, and water, oz. 40, three tablespoonfuls taken, iced, every hour, and he finds that it checks gastric irritability and

increases the secretion of urine. As a prophylactic quinine is useless. On the whole then, careful hygienic conditions, and the use of supporting measures when necessary, will afford the greatest hope of success in the treatment of yellow fever. The following measures should be adopted immediately the disease shows itself in a barrack or town :—

1. Complete isolation of those attacked. Evacuate the barracks.

2. Perfect sewage and ventilation.

3. Good water, freed from possibility of contamination.

4. Patients are best treated in the open air if the season of the year permit.

5. Remove all discharges and vomited matters at once, and mix them with chloride of zinc. Disinfect all clothing of infected persons. In ships the bilge-water should be pumped out, and the vessel fumigated. With regard to disinfection, fumigations with nitrous acid gas are said to be the only successful means at present known.

6. Rigid quarantine. This is of doubtful efficacy. It must be borne in mind that in this disease, as in many others, persons not affected may convey the fever to others. It is not necessary that those conveying the disease be themselves affected.

DENGUE.

Lat. *Denguis*. Fr. *Dengue*. Ger. *Dengue*.

DEFINITION—To a contagious disease, occurring epidemically, characterised by pyrexia, accompanied with excruciating pains in the head, small joints, muscles of the body and extremities, and with the presence of an eruption at one time like that of scarlet fever, at another like that of measles or urticaria, the name Dengue has been given.

Of this disease little is known, the first mention we have of its appearance being an account of the outbreak which

occurred among a body of troops at Rangoon in 1824. It has been called by some *breakbone fever* or *dandy fever*, and by others *scarlatina rheumatica*. Epidemics have occurred in the West Indies in 1827, and in several parts of the United States, chiefly in the Gulf States. In Europe the only place where the disease has been seen is Cadiz, in 1867.

Etiology.—It is a tropical or sub-tropical disease, probably caused by a specific organism which has not yet been discovered. It attacks all members of a community equally; but as it is never fatal nothing is known of its pathological changes.

Symptoms.—Those present in all fevers — hot skin, quick pulse, thirst, &c. The period of incubation is from three to five days. The onset of the disease in some cases is insidious, in others sudden. The most important feature of the disease is the terrible rheumatic-like pains in the joints, which are so distressing to the patient. They have a boring or breaking character, hence the name breakbone fever. The joints become swollen, red and painful, but effusion into them does not, as a rule, occur. The temperature may vary from 102° to 104° , and the pulse from 80 to 140, the fever generally reaching its height by the third or fourth day. During the nonfebrile condition which follows he feels weak and stiff. Delirium is rare in spite of the high fever and severe pain. In many of its symptoms it appears to be allied to acute rheumatism, scarlet fever, ague, and measles. Relapses, accompanied by a *second* appearance of the rash on the palms of the hands, the feet, and knees, are not infrequent, an attempt at convalescence being suddenly interrupted by a return of all the symptoms. Convalescence may be prolonged for three months, the joints remaining stiff for a considerable time.

Treatment.—The treatment consists in the administration of analgesics, such as antipyrin or phenacetin or even opium, to relieve the suffering, and the application of rubefacients to the spine and painful parts. Blisters may be of use over affected joints. Alcohol is often indicated, with light but nourishing diet and rest in bed. During convalescence, which is often tedious and prolonged, tonics—quinine and iron—and change of air are required; while potassium iodide, together with massage and sulphur baths, are of undoubted service for mitigating the pains.

DYSENTERY.

Lat. *Dysenteria*. Fr. *Dysenterie*. Ger. *Ruhr*. Syn. *Dysenterie*.

DEFINITION—Dysentery is a clinical term meaning inflammation and ulceration of the mucous membrane of the large intestine, most prevalent in hot climates, and often occurring as an epidemic.

Dysentery is supposed by some to be due to exposure to sudden and great variations of temperature *e.g.*, “a chill,” especially when the system is weakened by fatigue, irregular living, combined with insufficient clothing and lodging. Foul drinking water also has frequently been blamed. Malaria is held by some to be a cause of dysentery, and the frequent association of the two diseases must not be forgotten.

Pathology and Morbid Anatomy.—Dysentery has been divided into three forms—*catarrhal*, *diphtheritic*, and *amoebic*, from, in the last form, the presence of the amoeba coli, a unicellular protoplasmic motile organism found in the stools; but the first is merely a milder form of the second. This classification of dysentery is very imperfect.

The amoebic variety is perhaps the best known. Other varieties are supposed to be caused by—(1) The common bacteria of inflammation and suppuration; (2) the bacillus coli communis; (3) a specific bacillus; but the bacteriology is still undetermined. The inflammation of dysentery is most marked in the descending colon and rectum, but it may extend into the ileum. The rectum is believed by many to be the primary seat of the disease. It is characterised by swelling, ulceration, and sloughing of the mucous membrane of the bowel, varying, however, with the intensity of the inflammatory process. In the milder forms of the disease, the summits of the folds of the mucous membrane are the portions which show the morbid changes most. A greyish-white layer of fibrinous material is found covering them, which leaves, when scraped off, a slightly ulcerated surface. The solitary glands enlarge, slough, and thus produce ulcers, which increase in size rapidly. The ulceration is not confined to the solitary glands. The general mucosa is swollen, irregularly congested, and shows many small abscesses which rupture into the gut and form ulcers. In the severer forms of the disease the necrosis becomes intense, and large portions of the mucous membrane are converted into black, rotten sloughs, which, in the course of time, are thrown off by a sort of reactive, suppurative inflammation. The *dysenteric ulcer* is usually best seen on the ridges of the large intestine, with irregular undermined edges, the base being formed by any of the coats of the intestine. The ulcers are of all sizes, and frequently coalesce and may form irregular ragged areas extending throughout the entire length of the colon. Sometimes the ulcers may heal and recovery take place, considerable contraction resulting. In chronic dysentery the mucosa is darker and many cicatrices and indurated areas are seen. The gut is hypertrophied or thinned irregularly, and the lumen contracted and dilated. The amoebic variety is particularly apt to become chronic.

Symptoms.—Dysentery generally begins as a simple diarrhoea accompanied with some fever, but the temperature seldom exceeds 101°F . The important symptom of this disease is *tenesmus*, or the constant desire to go to stool, accompanied with great straining and pain, but inability to evacuate the bowel, nothing being passed but mucus, blood, and shreds of fibrin. Small hardened lumps of *fæces*, called *scybalæ*, are sometimes expelled, but, as a rule, all trace of feculence soon disappears, the discharges being thin, watery, reddish, turbid or dirty-looking—"meat washing"—or they may have the jelly-like appearance described as "boiled sago" or "frog's spawn." The excessive straining at stool often gives rise to a burning pain round the anus and to prolapse of the rectum, and the patient may even faint—the result of the intense prostration so characteristic of the disease. The griping or colicky pains which accompany an attack of dysentery are called *torminia*. In hot climates "tropical dysentery" is a very acute disease, attended with considerable febrile movement and intense pain around the umbilicus, as well as in the course of the rectum; but the symptoms of *tropical or amœbic dysentery* differ only in degree from those seen in sporadic dysentery of more temperate climates. The disease may end in—(1) Recovery; (2) death from collapse; or (3) in chronic dysentery. If the disease becomes chronic the stools may become fairly well formed, but jelly-like material and blood may accompany them, usually preceding the stools, which are irregular as to time.

The disease is not infrequently followed by hepatic abscess, both intra- and supra-hepatic, and the *amœba coli* and other micro-organisms are found in the pus.

Diagnosis.—Intussusception of the bowels, especially in male children, internal hæmorrhoids, cancer of the rectum, ulcerative colitis, arsenical poisoning, &c., might be mistaken for dysentery. Care is in all cases necessary.

The Prognosis in sporadic dysentery occurring in a temperate climate is favourable, but in hot countries it is a grave disease, and any opinion should be given cautiously. When malignant characters set in early a very grave prognosis should be given and a permanent cure is very unlikely in chronic dysentery of over two years' standing.

Treatment.—Diverse modes of treatment have been recommended for the cure of dysentery. Purgatives, calomel, castor oil and other remedies have all had their votaries. The plan at one time generally adopted in India, and found efficacious, consisted in the exhibition of large doses of ipecacuanha. The mode of its administration is as follows:—Half-a-drachm of the tincture of opium may be given followed in about a half-hour's time by 20 or 30 grains of powdered ipecacuanha in a little syrup. The patient is kept perfectly quiet, and he is not allowed to take any fluid, but he may suck small pieces of ice. In four or five hours the dose may be repeated. Nausea and vomiting rarely occur when drinking is prohibited. Irrigation of the bowel is one of the best and most rational modes of treatment for amoebic dysentery, although the intense irritability of the bowel and the terrible tenesmus induced renders the treatment difficult in the earlier stages, where it is most likely to be serviceable. Large injections must be used, of 20 to 30 grains of silver nitrate to the pint of water, and at least 2 to 3 pints slowly injected, or a solution of quinine, 1 in 2000 parts of warm water, may be substituted. Argyria is rarely produced by the silver nitrate injections. Small warm opium and starch enemata (30 to 60 minims of laudanum in 1 to 2 oz. of thin starch) are of very great benefit for relieving tenesmus. In chronic dysentery bismuth combined with Dover's powder may be tried, and salol is also useful. Morphia suppositories, with 1 to 2 grains of extract of ergot in each, have been found by us

to be of very special benefit in all forms of dysentery, and especially the chronic form. But perhaps the most important part of the later treatment consists in removing the patient to a suitable climate and in carefully dieting him. The diet should be chiefly composed of milk and farinaceous foods, and the patient should be warmly clothed and have complete rest, both of mind and body.

BERI-BERI.

Syn. *Kakke*.

DEFINITION—A specific form of multiple peripheral neuritis, characterised by, in addition to the usual sensory and vasomotor disorders of such neuritis, a special implication of the phrenic and vagus nerves and a liability to general dropsy or atrophy.

Etiology.—It prevails particularly in Japan, Brazil and the Dutch East Indies, but it is also met with in many other countries, and though most common in tropical, it is also found in temperate regions. There was a marked outbreak in Dublin during 1896, 1897, and 1898. It is believed to be due to a germ, and several different organisms have been described, but no definite pronouncement can yet be made. Nor can we say whether the poison is produced within the body or outside it, say in the soil. Overcrowding, heat, damp and a diet deficient in nitrogen, as well as malaria and other weakening influences, are strong predisposing causes; but it is essentially a locality disease, and clings with great tenacity to a region, place or building which has once become infected, so that newcomers taking up residence therein readily become infected.

Pathology.—There is a constant change in the muscles and nerves. The nerves supplying the affected muscles are

degenerated, the extent of the change being most marked at their peripheral ends, and diminishing as they ascend upwards to the cord. The same degenerative changes are seen in the phrenic and vagus nerves, in the laryngeal branches of the latter, and in the nerves of the heart. The muscles first give the reaction of degeneration and then become paralysed. The most constantly paralysed muscles are those supplied by the external popliteal nerve, and the next most frequently are the flexors of the toes. In the arm it is the extensors of the hands and fingers. The diaphragm, abdominal and intercostal muscles become affected, and the laryngeal muscles are frequently involved. It is this affection of the respiratory muscles which often causes death. Microscopically, the affected muscles show marked granular degeneration and atrophy. The heart is prominently affected; its chambers, particularly on the right side, being dilated, and its muscular fibres showing granular degeneration and foci of leucocyte infiltration. The posterior roots of the spinal nerves show atrophied fibres between the ganglion and the nerve trunk. There is often marked and general dropsy affecting all the tissues and serous cavities, the pericardium particularly, either along with the marked atrophy above mentioned, or when it is but slight. It is probably due to the dilatation of the heart and the vaso-motor disturbances.

Symptoms.—There is often a premonitory period in which the patient feels at times tired and depressed, with a feeling of numbness or cramp in the legs, and shows a swollen puffy face and some dropsy along the legs or ankles. Then definite symptoms of peripheral neuritis come on slowly or rapidly. The calves of the legs show an area in which tactile and electrical sensations are lost, and are painful when pinched. The muscles become weaker, so that he rises with difficulty, wavers when he stands with his eyes closed, and walks with difficulty. In raising the foot,

the toes leave the floor last, then the foot is raised higher than usual, and after being pushed forward falls suddenly with a flop on the floor again. The œdema and weakness increases, and the anæsthesia in the legs extends and then appears also in the thenar and hypothenar eminences of the hand; the knee jerk completely disappears. The affected muscles, particularly those supplied by the external popliteal nerve, give the reaction of partial degeneration. The faradic current elicits no response, and there is also diminished irritability to the galvanic current. This reaction of degeneration is an early symptom, and persists throughout the disease. Digestion is good. Death, which is frequent, may be due to heart failure or to paralysis of the respiratory or laryngeal muscles. Recovery may set in after a variable time of ups and downs through a gradual abatement of the symptoms, and in three months or so, sometimes much longer, he may be able to go about and resume work.

Prognosis.—It is a serious disease, the mortality being about 20 per cent. Marked cardiac, dropsical, respiratory mischief or vomiting are unfavourable signs.

Treatment.—The prophylactic measures which should be adopted to arrest the spread of the disease are dieting—replacing the purely rice diet with fresh meat, vegetables and milk—preventing overcrowding and attending to sanitary conditions.

During an acute attack saline purgatives may be administered from time to time with great benefit, the heart stimulated when necessary with digitalis, strophanthus, or other cardiac tonic, and the paroxysms of dyspnoea treated when they appear with inhalations of nitrite of amyl (3 minim capsules), nitroglycerine, and sometimes by venesection. Salicylates have been recommended as being of great value in the treatment of the disease.

When local pain has ceased, the wasted muscles should be treated by massage, electricity and strychnin hypodermically. Pleural and other effusions if excessive must be tapped. When at all possible, remove the patient from the area of the disease.

SUB-GROUP (e)—*INFECTIVE DISEASES COMMUNICABLE FROM ANIMALS TO MAN.*

PLAGUE.

Lat. *Pestilentia*. Fr. *Peste*. Ger. *Pest*.

DEFINITION—A specific, highly infectious and contagious fever, attended with buboes of the inguinal or other glands.

Etiology.—Plague is caused by a specific bacillus discovered by Kitasato and by Yersin independently in 1894. It is a short oval bacillus with rounded ends, and non-motile, though possessing flagella. The ends often stain deeply, leaving the central part unstained. This is spoken of as polar staining. It may enter the body of man—(1) Through abrasions on the skin, (2) through the respiratory and (3) the alimentary systems. It attacks lower animals also, and rats and mice die in great numbers from it in an affected area. The rat plays an important part in the transmission and spread of the disease. They may be infected by contaminated soil or clothes or by eating contaminated grain or the carcass of an infected fellow rat. It is not yet definitely known how the infection may be transmitted from the rat to man, but enough is known to justify the extermination of rats in a contaminated area. It has even been suggested that the disease is a primary one in rats and only secondary in man, but this lacks proof. In some outbreaks man has been affected before the rat and *vice*

versa, and in others the affection has seemed to be simultaneous. Manson puts it tersely thus, for the prevention of cholera the tea-kettle, of malaria the mosquito net, and of plague the rat-trap. Thus, mosquitoes and other parasitic insects may convey the poison. It may also be conveyed by fomites or by the atmosphere of the sick-room; but this may easily be avoided by free ventilation.

Plague has occurred in Europe in several severe epidemics in the past, the last great one being the Great Plague of London in 1665, when nearly 70,000 persons died. In recent times epidemics have broken out in China and in India, where it has now been rife since 1897. A comparatively limited outbreak occurred in 1899 in Oporto, and two still milder ones in 1900 and 1901 in Glasgow. Sporadic cases have occurred at different places occasionally, so that the disease may be now truly regarded as pandemic.

Pathology.—The affection of the lymphatic glands is the most striking feature of the disease—those of the groin most frequently (about 50 per cent.), those of the axilla next (about 30 per cent.), and, lastly, those of the neck (about 15 per cent.). It is believed that none of the lymphatic glands escape, but the affection is most marked in those named. They are swollen, one gland being generally much more swollen than the others; and the surrounding tissues are oedematous and hæmorrhagic. On section the glands themselves look red in the case of the smaller ones, and pale pink with hæmorrhagic foci in the case of the large ones. The latter become softened and necrosed, but true suppuration is not common and is always a late feature, rarely appearing before the eighth or ninth day. One group of glands is usually affected first (the primary bubo), and later others become involved. The bacilli are present in great numbers in the swollen glands, and cover-glass films made from a scraping of the gland, or from a hypodermic needle plunged

into it, may show them abundantly and be of great importance in diagnosis. There is also great swelling of the spleen and cloudy swelling of the liver, kidneys, &c., and bacilli are often abundant in these organs. The most marked hæmorrhages are often seen around the kidney. This is commonly called the bubonic form of the disease on account of the prominence of the lymphatic gland enlargement. There is another form which mainly attacks the lung, called the pneumonic form, the glandular enlargements being slight or late features. It is a lobular pneumonia although large areas of the lung may become involved from the confluence of contiguous pneumonic foci. The individual foci themselves vary from the size of a pea to that of a hen's egg or larger. There may be little or no cough or expectoration, but when the latter is present it shows bacilli, often in enormous numbers—another important diagnostic test. In this form it is said to be always fatal. There is a third form, the septicæmic, in which death occurs rapidly without any obvious bubo formation. In all forms bacilli occur in the blood, though often not till late in the disease, and often so scantily that they may escape detection in films but may be easily recognised by cultivation. They also escape with the urine and fæces. The mild type of the plague is called *Pestis Minor*, and the severe type *Pestis Major*. Each may prevail separately to the exclusion of the other, and the latter may be developed out of the former. The recent epidemic, that of 1900, in Glasgow is a good instance of *pestis minor* and was mainly characterised by glandular swellings, slight fever, and the absence of severe general symptoms. Pneumonic forms may occur. The mortality in *pestis minor* is slight, but in *pestis major* it may exceed 90 per cent. and rarely falls as low as 30 per cent.

Symptoms.—The period of incubation is imperfectly known, but is generally three to five days, very occasionally ten

days. The onset is often sudden, or there may be headache, giddiness, languor, injected eyes, or vomiting for a day or so, often less, before the fever appears. This may be ushered in by a rigor and rises rapidly to 102° or 104° or even to 107° . The pulse is rapid, generally over 100. Constipation is the rule at first, often followed by diarrhœa later. There may be great apathy or violent delirium. The buboes appear usually after the onset of the fever—on the second, third, fourth, or even fifth day of the disease—and last during the whole of the attack. They are at first hard and painful, and afterwards soften. They do not generally exceed a walnut in size. The fever lasts usually from two to five days and generally subsides suddenly. The fourth and fifth days are the most fatal periods of the disease, though cases are sometimes fatal within a day. Necrotic patches, wrongly described as carbuncles, occasionally occur on the skin. When suppuration occurs in the buboes the cases, though generally regarded as favourable, may last three weeks or a month or even longer.

Diagnosis.—The first cases of an outbreak often escape notice, either because of their mildness or because of their occurring in the pneumonic form, as in Glasgow. This error might be partly avoided if the sputum in every case of pneumonia were examined microscopically as a routine procedure. When buboes appear the disease is characteristic enough, as no other acute febrile disease shows the same affection of the lymphatic glands, but the diagnosis should at once be made absolutely certain by microscopic examination, cultures, and animal inoculation.

Prognosis.—A guarded prognosis, even in mild and apparently favourable cases, should always be given. Hæmorrhages and petechial eruptions are very unfavourable signs, and so also is profound affection of the nervous system.

Treatment.—While much may be hoped for in the near future from serum therapy, the greatest care must be taken with the nursing, diet and stimulation of the patient. Constipation is often severe, and a single good dose of calomel has been recommended, especially as it is said to relieve the vomiting, and thereafter opium or morphia must, in the initial stages, be pretty freely used. Opium has been found to check the great restlessness, delirium and pain, and of course will procure sleep. Stimulants must not be neglected, and should be given from the outset, such as alcohol (in the form of iced wine, champagne or malt liquors), the spirits of ether, ammonia, chloroform, and strychnin. Never give any of the ordinary antipyretics, as they depress the heart, which is only too apt to fail in any case, and the cold sponging and other hydropathic means of checking hyperpyrexia are much safer and better.

Ice cream is of benefit and may be freely taken. The buboes require careful treatment and should first be smeared with belladonna extract and vaseline, and later treated as an ordinary boil and opened.

Lately a number of vaccines or serums have been tried: of these, Yersin's, Haffkine's, Roux's and Lustig's are all well known and have to a certain extent been successful. Haffkine's vaccine is a sterilised bouillon culture of the plague bacillus, and its value as a prophylactic has been abundantly proved, while the others are antitoxic serums, of which Yersin's is obtained from the horse; but their curative power has yet to be demonstrated.

The destruction of rats seems to have been even more efficacious than the most careful quarantine measures, necessary as these are. Absolute sanitary perfection is one of the best means of preventing epidemics and of limiting the disease when it is accidentally introduced into a community.

GLANDERS AND FARCY.

GLANDERS.

Lat. *Equinia*. Fr. *Morve*. Ger. *Rotz*.

DEFINITION—An infectious inflammatory disease of the nasal mucous membrane, caused by the bacillus mallei.

FARCY.

Lat. *Farciminum*. Fr. *Farcin*. Ger. *Wurm*.

DEFINITION—An infectious inflammatory disease of the subcutaneous tissue and of the lymphatics, caused by the bacillus mallei.

Glanders is a disease of the horse and ass, communicated occasionally to man and other animals. It is a rare disease in man and nearly all the cases are in males whose occupation closely associates them with horses—*e.g.*, grooms, stablemen, knackers, &c.

Etiology.—The exciting cause is a bacillus—the bacillus mallei—which closely resembles the tubercle bacillus, but is a little shorter and somewhat thicker than it. It both stains more easily and is more readily decolorised than it. It grows readily upon all the ordinary culture media, both aerobically and anaerobically, giving a characteristic growth on the potato. The poison usually enters through an abrasion or wound of the skin, but it may enter through the hair-follicles in an apparently unbroken skin, and it can be absorbed by an intact mucous membrane. The disease is transmitted from the sick to the sound, principally by the discharges from the diseased surfaces, and also by such excretions or secretions as urine or milk. It has been transmitted directly from man to man. It is highly contagious, and spreads in horses especially, often with great rapidity.

Morbid Anatomy.—The bacilli cause a local reaction in the tissues, producing nodular growths of the granulomatous type, generally comparable to those of tubercle, but with a greater tendency to break down and suppurate. There are collections of leucocytes and epithelioid cells, but giant cells are comparatively rarely seen. If the respiratory system is mainly affected the name Glanders is used, from the marked swelling of the lymphatic glands of the neck which usually occurs; if the lesion is mainly in the skin the term Farcy is employed, but no hard and fast line can be drawn between the two forms, either clinically or pathologically. Acute and chronic glanders, and acute and chronic farcy are spoken of according to the rapidity of the process.

Symptoms.— The disease sets in after a variable period of incubation, of from three to five days usually, but sometimes longer, with malaise, rigor, fever and pains, especially about the joints and in the muscles. Swellings appear at the site of inoculation, either beneath the skin, where they are more prominent, or the nasal mucous membrane or elsewhere. There is general redness and lymphangitis. The swellings rapidly break down and form suppurating ulcers. Similar swellings rapidly appear in other parts of the body. Papules, which rapidly become pustules, may appear in the face and be for a time the only lesions seen externally, thus resembling small-pox. When the nasal mucous membrane is affected there is a discharge, at first ichorous and mucoid and soon becoming purulent; but in many cases it is little if at all affected. In the skin the disease spreads by the lymphatics, prominent swellings (the farcy buds) forming at intervals, and rapidly suppurating similar nodules are seen among the muscles in the lymphatic glands, and in the lungs and all the internal organs. The lungs are the organs most frequently affected; miliary foci, never so numerous as in tubercle,

lobular pneumonic foci, larger pneumonic areas, and interstitial pneumonic patches being the more frequent forms of lesion. Suppuration is the most constant feature in all the lesions, whether external or internal. The acute cases last about a fortnight; the chronic cases may be very prolonged, even to many months, causing chronic foetid forms or deep external ulcerations, with thickened lymphatics and swollen glands, or intra-muscular abscesses of an untractable and ill-defined character. These cases may at any time become acute.

Diagnosis.—It is often difficult and should be cleared up by bacteriological examination of the pus or the discharges, by making cultures therefrom, and by inoculations into the peritoneum of a guinea-pig, when a glanderous caseation of the testicles will supervene in positive cases in from two or three days to a week or more. The agglutination test is also of value, and in animals mallein is used with great success.

Prognosis.—Acute cases nearly all die. Of the chronic cases about 50 per cent. are said to recover.

Treatment.—The chief thing is prevention, and a wound or bite which is probably infected should be cauterised or excised. Mercury has been tried, but with little success. Iodide of potassium may be given; and the inhalation of creosote is useful as an application to the inflamed nasal passages. Injections of carbolic acid into the nose may be tried. As a rule, however, the disease, especially if acute, is fatal in spite of treatment. When the disease occurs in the horse, the animal should be shot, the stables should be disinfected, and not again used for a year or more.

ANTHRAX, OR SPLENIC FEVER.

CHARBON, SPLENIC APOPLEXY, MALIGNANT PUSTULE, WOOL-SORTER'S DISEASE.

Lat. *Febris splenic.* Fr. *Charbon.* Ger. *Milzbrandfeber.*

DEFINITION—An infectious widely-spread disease, attacking cattle and other herbivorous animals, and more rarely horses, occasionally infecting man, often occurring epidemically, caused by a specific bacillus.

In the lower animals the spleen is greatly enlarged, congested and softened, hence the name splenic fever; but in man the spleen usually shows little change and the term anthrax has come to be more extensively used.

Etiology.—The bacillus anthracis is the exciting cause. It is a large, straight, non-motile, spore-bearing bacillus, which grows readily upon all ordinary media, both at room and incubation temperatures. The spores require an abundant supply of oxygen and other conditions for their development, and hence they are not found within the living body nor in the unopened carcase after death. They are extremely resistant to injurious influences and retain their vitality in the soil for very long periods. The contagion usually enters the body of man by inoculation, through an abrasion of the skin or the hair-follicles, or by inhalation of the spores. It is also said that the disease may be contracted through the ingestion of contaminated food or milk. When the bacillus has entered the body it multiplies rapidly at the seat of inoculation, but remains localised, being confined to the pustule and its immediate neighbourhood, and does not enter the circulation for several days, generally four at least, but often more. After this it enters the circulation and is found in immense numbers in the blood and in the capillaries everywhere throughout the body. It becomes, therefore, an excellent example of a typical septicæmic

disease. The persons who are liable to the disease are those who handle hides and hair—*e.g.*, tanners, hide porters, wool sorters, butchers, knackers, shepherds, &c. It is very rarely communicated directly from person to person. In the lower animals it is commonly contracted through the spores by feeding in infected pastures or fodder or water. The bloody excretions from the mouth, nose, and bowels of infected animals contain the bacilli in immense numbers.

Morbid Anatomy.—It occurs as an external and an internal form, somewhat after the fashion of glanders. The external form is known as the malignant pustule, and the internal affects the lungs (wool-sorters' disease) or the intestine. The malignant pustule is generally seen on the face or neck or other exposed parts of the body, such as the hand or arm. In hide porters it is commonly on the back. It begins as a papule, which becomes a vesicle, usually surrounded by an area of great congestion. The vesicle, which may vary in size from a split pea to a shilling, ruptures and dries up, leaving a red, swollen, irritable and somewhat painful sore, the floor of which becomes necrosed and gets dark and unhealthy-looking, and about the third day or earlier shows a characteristic appearance. In the centre there is a dark brown or black depressed slough, generally surrounded by a raised ring of small red vesicles, outside which is an extensive area of inflamed skin and subcutaneous tissue, at first firm and oedematous, but soon becoming brawny and indurated. Serous fluid exudes, in which anthrax bacilli are generally found. The inflammation spreads along the lymphatics, and the neighbouring lymphatic glands are enlarged and inflamed. In favourable cases the slough separates about the sixth or seventh day after inoculation and the wound gradually heals, but in most cases when left alone the bacilli have become generalised by the fourth day or later, and the toxæmia increases. Occasionally in very grave cases there is no

malignant pustule, but a general erysipelatous œdema, which may result in gangrene.

Wool-sorter's Disease.—In this country it is generally restricted to the wool-sorting warehouses of Bradford and district. Lately a few cases have occurred in Kidderminster. The mucous membrane of the trachea and bronchi shows swollen, congested, often hæmorrhagic patches, but rarely necrosis. These lesions show numerous bacilli, as also do the bronchial glands, which are greatly enlarged, and the œdematous, often hæmorrhagically infiltrated, mediastinal cellular tissue. In intestinal anthrax there are similar lesions in the intestine and mesenteric glands.

Symptoms.—Coincident with the appearance of the malignant pustules there is generally fever, which becomes more marked in proportion as the sloughing and induration become more pronounced, but falls in the milder cases with the separation of the slough, or in the severe ones with the onset of collapse. It is often accompanied by prostration and depression, rigors, and sometimes vomiting and delirium. Death, which is the usual result, may be due to œdema of the larynx, or more commonly to the toxæmia.

In wool-sorter's disease the onset is usually sudden. The patient feels very ill, complains of pains in the back, of chilliness, and of oppressed breathing. There are rigors and considerable fever at first, but as collapse comes on the temperature falls to or below the normal. The pulse is rapid and feeble. There are usually no physical signs in the chest except those of acute bronchitis. The sputum may be blood-stained and contain the bacilli. Death may supervene from collapse in several hours, but those who survive for a week generally recover.

In intestinal anthrax the symptoms are similar, and diarrhœa, often hæmorrhagic, is frequently pronounced. The symptoms are generally those of intense poisoning.

Diagnosis.—In the external form the characters of the malignant pustule generally make the diagnosis easy. In the internal form it is often very difficult. The occupation should be enquired into. All the discharges, and in the later stages the blood, should be repeatedly examined bacteriologically for the bacilli.

Prognosis.—In the external form it is not nearly so grave as in the internal form, which is nearly always fatal. If the local lesion be on the head or neck it is much more serious than upon the extremities. It is greatly improved by early free excision of the lesion.

Treatment.—In man the treatment consists in the early destruction of the pustule with chloride of zinc or strong liquefied carbolic acid, or the hypodermic injection of a 10 per cent. solution of iodoform in ether. For the internal form of anthrax little can be done. Tonics and a supporting diet should be given. Quinine internally in large doses has been found useful.

RABIES--HYDROPHOBIA.

Lat. *Hydrophobia, hyssa*. Fr. *La rage*. Ger. *Hundswuth*,
Wassenschen, Tollwuth.

DEFINITION.—An acute infective disease produced in the human subject from the bite of a rabid animal.

Etiology.—1. ANIMALS AFFECTED.—It is most frequent in dogs, but other carnivora, viz., wolves, cats, and foxes, are not infrequently affected. It has occasionally occurred in horses, more rarely still in cows and pigs, and deer may also contract it. It can be inoculated into all warm-blooded animals, particularly rabbits, guinea-pigs, and monkeys.

2. GEOGRAPHICAL DISTRIBUTION.—It is most common in France and Russia and the East. It is rare in Germany, and is unknown in many regions—*e.g.*, Norway and Australia. Owing to the enforcement of the muzzling order it has practically disappeared in Great Britain, although in 1894 there were 248 cases, and in 1895 672 cases in dogs alone.

3. THE VIRUS.—The exact nature of the poison is still unknown, though all the facts point to its being due to some organism which can multiply in the tissues. The virus is at first found in the medulla only, later it is distributed throughout the whole nervous system, both central and peripheral, being more virulent in the former than in the latter. It is secreted chiefly by the saliva (especially the parotid), but also by the mammary, lachrymal and pancreatic glands. It is not present in the blood, tissues, urine or other excretions. It has been found in the saliva of the dog three days before the symptoms of rabies began to appear. Heat, sunlight, drying and antiseptics, *e.g.*, carbolic acid, are all capable of destroying it comparatively easily. Putrefaction does so more slowly.

4. METHOD OF INFECTION.—The poison usually enters the body in the saliva by a bite from a rabid animal, though it may do so through a healthy mucous surface, *e.g.*, conjunctiva and nasal mucous membrane, and possibly also be transmitted from the mother to the foetus.

5. METHOD OF SPREAD.—It apparently spreads from the site of inoculation by the path of the peripheral nerves to the central nervous system, and the rapidity and severity of the attack depend to some extent upon the ease with which this occurs. Thus slight superficial wounds, in which presumably a small amount of poison is introduced, not in direct contact with a nerve, do not usually give rise to symptoms until after long intervals, while deep and lacerated wounds do so much more speedily and severely. Similarly, bites upon the face and hands and exposed parts of the

body are more serious than those through clothes, because much or all the poison may be rubbed off before the teeth break the skin. Hence in man only about 16 to 25 per cent. of those bitten by rabid dogs contract the disease, males and children more commonly than women being victims.

6. PERIOD OF INCUBATION.—In man, it is usually about six weeks, but it is often very variable. In a few cases it is described as being less than three or even two weeks, and sometimes as long as one or two or even five years. In animals it varies with the species, ranging in dogs usually from fifteen to sixty days. In the rabbit it is usually fourteen to nineteen days. Apart from the species the chief cause of variation is the degree of virulence of the poison. Ordinary street rabies varies greatly in virulence and in period of incubation, but the virulence of laboratory rabies can be either greatly exalted or attenuated, and a virus can be obtained (the virus fixe of Pasteur) in which the incubation period is reduced to seven or even six days, at any rate for rabbits. This is the strongest virus which has so far been obtained, being stronger than that which is got from rabid wolves.

Symptoms.—IN THE DOG.—The most common clinical type is "furious rabies." The dog becomes sullen and morose, and hides in quiet corners. It then becomes restless but is not savage. It snaps at anything which comes in its way, but does not show any tendency to attack persons. The restlessness increases and it may wander long distances. The conjunctivæ become injected and the saliva increases and becomes sticky and frothy. It has no fear of water, which it takes readily, hence the term hydrophobia is not applicable to the disease in dogs, and rabies should be used. The bark becomes prolonged and high pitched. The excitement increases until the animal rapidly becomes furious and maniacal, attacking everyone

and everything that comes in its way. It bites at and swallows all sorts of foreign materials, such as small stones, bits of wood, bone, hair, hay and straw, coal, its own fæces, &c. Emaciation is rapid, and after two or three days paralysis sets in, affecting first the lower jaw and the hind limbs. There is generally some difficulty of swallowing, especially liquids, even during the restless stage. The paralysis extends rapidly to the rest of the body and the respiratory muscles, causing death by asphyxia. The disease may run its course in two days, but generally lasts four or five, and sometimes even ten days. There is another clinical type, the "dumb or paralytic" rabies, occasionally seen in the dog. It is the characteristic type in the rabbit, and may occur in man. The disease is more rapid, the paralytic stage rapidly coming on after a slight and short period of excitement.

IN MAN.—He may first complain of a tingling or pricking feeling or a sensation of heat or sharp pain in the region of the wound. He becomes anxious and depressed and complains of being unwell. Sleep becomes broken. He is quite rational, but irritable. He can talk freely, but his respiration being slightly interfered with, his sentences are sometimes interrupted. There is much thirst, and he feels disinclined to swallow. The pulse becomes quickened. On the second or third day he generally passes into the stage of excitement. He becomes very restless and agitated and hyperæsthetic. Slight intermittent delirium may intervene. Speech becomes disconnected. The mucous membrane of the mouth and fauces becomes congested and a thick tenacious mucus accumulates, causing a harsh coughing in his efforts to get rid of it. The thirst increases, and he makes strong efforts at first to assuage it with water, but so hyperæsthetic has the throat become that the attempt causes violent spasmodic contraction of the muscles of deglutition, resulting frequently in the fluid being expelled from the mouth. Soon the spasm passes

to the muscles of respiration and the other muscles of the body, causing a general tetanic condition with marked opisthotonus. These attacks recur at every attempt to swallow, so that very soon the mere sight or suggestion of water is terrifying to the patient, hence the name hydrophobia. The hyperæsthesia increases, so that even slight reflex stimuli, such as a bright light, or a loud sound, or a draught of cold air, may set up a convulsive attack. The mental excitement of the patient increases, and maniacal symptoms may supervene during the convulsions, although the mind is unclouded in the intervals when the patient is quiet. The temperature may remain normal, but it is sometimes slightly increased to 100 or 101°F., and often rises to 103 or even more during the convulsive attacks. These attacks usually increase in frequency and severity, and the patient dies asphyxiated in from two to four days. Occasionally death is due to exhaustion. The stage of excitement is sometimes so evanescent that the first symptoms seem to be those of a rapidly-ascending paralysis resembling Landry's paralysis, and it is possible that some of the cases described under this disease may really have been very rapid examples of this form of hydrophobia.

Morbid Anatomy.—There is congestion of the pharynx, larynx, trachea, stomach and other parts of the alimentary canal. There are also changes in the nerve-cells, the blood-vessels and their perivascular lymphatic sheaths in the spinal cord and medulla, especially the latter, which most authors, however, do not regard as specific, so that it is very difficult to diagnose rabies from a postmortem examination alone. Babes and a few other authorities, however, claim that this can be indubitably done, since they regard certain of the spinal cord changes as absolutely pathognomonic.

Diagnosis.—It is of the first importance that the earliest possible diagnosis should be made in every case. By

isolating and observing the dog, a fairly certain opinion may be obtained. The dog should not be killed unless the symptoms are indubitable, as death will generally follow soon, the average duration of the disease being usually four or five days, and the whole range of symptoms generally enable a clear diagnosis to be made. The head should then be carefully packed and sent to a scientific laboratory if the observer is not himself able to carry out the investigation. The medulla is placed in glycerine for two or three days. An emulsion of it is then made in sterile salt solution or broth and injected subcutaneously, or into the muscles or the aqueous humor, or best of all, subdurally in the rabbit; the spinal cord may be selected instead of the medulla. In positive cases—*i.e.*, if the disease be rabies—the rabbit develops characteristic symptoms of paralytic rabies (progressive paralysis with attacks of chronic spasm and dyspnoea) in from fourteen to nineteen days, though the incubation period is sometimes prolonged to six weeks. Death usually follows in two or three days. The clinical course of the disease in dogs is the quickest method of diagnosis, and enables the specific treatment to be begun early.* The diseases most likely to simulate rabies are epilepsy, epileptiform attacks, and enteritis. A foreign body in the throat may cause a mistaken diagnosis of dumb rabies. The inoculation method is more certain, but it may take several weeks. When the clinical symptoms have appeared in man the diagnosis is not often difficult. There is sometimes a difficulty in cases of mania, and when a fatal termination supervenes the medulla or spinal cord may be used in the same way as in dogs, &c., to clear up the diagnosis. A hysterical train of symptoms, termed

* It has been recently shown by Negri and others that the motor cells of the brain, particularly of the cornu of the hippocampus major, contain rounded, oval or irregular bodies varying in size from 5 to 20μ and having a hyaline protoplasm, an inner body (nucleus?), and granules. Their presence can be easily detected in smears or sections, and being specific for the disease enable a rapid diagnosis to be made. They are thought to be protozoa and are claimed by some to be the causal germs of the disease.

pseudo-hydrophobia or lyssophobia, is sometimes seen in nervous persons bitten by a dog suspected of rabies. The patient is very frightened and depressed, suffers from difficulty in swallowing, and even general convulsions. There is, however, never any true respiratory spasm or rise of temperature. It does not progress, and lasts much longer than the true disease, being also amenable to treatment.

Prognosis.—It is difficult to arrive at even an approximately true percentage of the number of deaths in those bitten by rabid animals before the introduction of the Pasteur treatment. It is variously stated to be from five to eighty per cent., probably the most trustworthy percentage is sixteen. The patient almost invariably dies within four days after the commencement of the symptoms. Now that the Pasteur treatment has become general the prognosis is excellent.

Treatment.—The destruction of stray dogs and the muzzling of the rest during warm weather are almost sufficient to stamp out this disease, and these measures have greatly reduced the number of cases of canine rabies. When a dog supposed to be mad has bitten a man the animal should, if possible, be caught and kept alive, and prompt attention paid to the bite. Where the bite is through clothes, much of the danger from the infective saliva is removed, because the teeth either never come in contact with the patient's skin or are wiped fairly clean. A bite on exposed skin should be carefully cleansed, and thereafter cauterised by strong carbolic or fuming nitric acid, or the part may even be excised should the case come under early observation. If the animal is saved alive and develops rabies the patient must at once be treated by the Pasteur method. In brief, the principle of the method is this;—The virus inoculated

from the brain of a mad dog into a rabbit produces rabies in fourteen to nineteen days. A second rabbit inoculated from the first acquires rabies in a shorter time, and in successive inoculations in rabbits it is found that the virulence of the virus increases rapidly. The so-called *virus fixe* is that obtained from a rabbit rendered mad in seven days. The spinal cords of these rabbits contain the virus, and when they are carefully dried in the air the virus gradually diminishes in virulence. A preparation made from rabbit's cords suspended for fifteen days is the weakest, and therefore the first used for the inoculation of patients, and at stated periods inoculations with cords suspended for shorter and shorter periods are administered until the patient is immune to the *virus fixe*, and he will therefore be capable of resisting the virus if it develops following on the bite, whether of a rabid dog or cat. The inoculations are administered subcutaneously and generally into the abdominal wall. They are somewhat painful and often cause some constitutional disturbance.

Should hydrophobia actually manifest itself in man, the prognosis is most unfavourable. The patient must be kept in a darkened room and absolutely quiet. Chloroform is frequently needed to allay spasm, and cocaine painted on the throat may enable the sufferer to swallow nourishment. Nutrient enemata are of value, and all kinds of sedatives, such as opium, chloral and bromides, may be tried.

THE FOLLOWING TABLE IS TAKEN FROM HOLMES'S
"SYSTEM OF SURGERY."

HYDROPHOBIA.	TETANUS.
GENERAL CHARACTER.	
<p>Spasms of muscles of brief duration; if not voluntary, at least temporary, and ceasing to exist during intervals of rest and quietude, the jaw being relaxed, and opening and shutting regularly. The spasms are clonic.</p>	<p>Spasms of muscles more continued, less remitting, and more intermitting; constant rigidity of the muscles of the jaw, becoming gradually fixed and closed; tonic spasm. The cause is exposure to cold, or a wound; it rarely arises from the bite of an animal, and generally occurs soon after the injury. The bite of a tetanic animal does not produce tetanus.</p>
<p>Vomiting and gastric pains general; mind subject to rabid influences and numberless deviations, passing to delirium; intolerant sensibility of surface and organs of sense.</p>	<p>Vomiting and gastric pains rare; mind generally clear to the last.</p>
COUNTENANCE.	
<p>There is an expression of excitement, fearful distress, and peculiar restlessness, occasionally frightful convulsions; the eyes are bright and glistening, but at times suffused; thirst and aversion to fluids characteristic, even the sight or noise or fluids induces paroxysms, with frequent and viscid discharge of saliva.</p>	<p>Drawing up of the nose, wrinkling up of the forehead, angles of the mouth drawn towards the cheek-bones, presenting a frightful risus sardonicus. There is an expression of pain, but the eyes are natural; no great thirst, and in general no great aversion to fluids administered in small quantities; rarely any discharge of saliva.</p>

FOOT-AND-MOUTH DISEASE.

DEFINITION.—An acute infective disease of ruminants and pigs chiefly, though communicable to other animals, such as horses, dogs, cats, and fowls, and to human beings.

Etiology.—It is in all probability due to a specific micro-organism, and Klein, Schottelius, Behla and Kurth have each described a germ which they regard as the responsible cause. All these observations lack corroboration. It has been recently stated that the germ of this disease, as of African horse sickness, pleuro-pneumonia, &c., is so small that it passes through a porcelain filter, and hence it is too small to be detected by the powers of our present microscopes. It multiplies only in the bodies of affected animals. Insects, such as flies, are thought to carry the infection. Contact between animal and animal is the commonest method of spread, but places, persons, animals and things may become merely contaminated with the poison, and originate fresh outbreaks. The virus is abundantly present in the vesicles which form on the mucous membranes or skin, and hence the buccal saliva becomes highly charged with it.

Pathology.—Vesicles varying in size (from a sixpence to a five-shilling piece in the cow) are found on the mucous membrane of the mouth and skin of the feet, hence the name given to the disease. After bursting they form shallow ulcers; subsequent inflammatory changes of any severity are due to the entrance of adventitious organisms, which are naturally present in the affected regions, particularly the feet. Vesicles may also appear in other situations, *e.g.*, the teats, nostrils, throat, &c. In the human subject,

to whom it has frequently been transmitted, either through affected animals or milk, the vesicles are similar.

Symptoms.—The animal loses its appetite and refuses to feed, and frothy saliva escapes from its mouth. It frequently moves its feet. The vesicles appear within a few hours or so of the onset of the illness. In sheep the foot lesions are very prominent, while the mouth lesions are slight. In the human subject the mouth is most frequently and mainly affected, but the hands are also often affected, while occasionally vesicles appear also on the nose, breasts, &c. The disease is generally mild, complete recovery taking place in about ten days.

Treatment.—When the disease is transmitted to man, its treatment must be on general lines. The mouth should be washed out with antiseptic solutions, such as permanganate or chlorate of potash, the alimentary disturbances treated as they arise, and the general health and strength of the patient maintained.

It is rare that the disease proves fatal; but every care should be taken to prevent its spread, and it is hardly necessary to add that kissing on the mouth should be strictly forbidden, while the infected milk or butter should on no account be used as food.

SUB-GROUP (θ)—*DISEASES DUE TO PROTOZOA.*

MALARIAL FEVER.

DEFINITION—An infectious disease caused by the hæmatozoon or plasmodium malarie, and characterised by fever of (1) intermittent type—intervals of pyrexia and apyrexia regularly alternating; (2) remittent type—where the fever has marked remissions or slight remissions (continuous). It is now known that the old distinction between intermittent and remittent fevers no longer holds good pathologically, for both are varieties of the one disease—malarial fever or paludism.

Etiology.—1. GEOGRAPHICAL DISTRIBUTION.—It is most prevalent in tropical countries. It is rare in temperate regions but increases progressively towards the equator. It is rare in Europe, except in Southern Russia and Italy. It is not uncommon in certain parts of America, mostly isolated, however, in its distribution. It is very prevalent in many parts of India and Africa, particularly in low-lying districts and in the deltas of large rivers.

2. SOIL CONDITIONS.—It has been well named a "soil" disease. Sailors in malarial regions are said not to be attacked unless they land. Marshy land and stagnant water favour its development, and its disappearance from such places after proper drainage, &c., has been attributed to these sanitary improvements. An outbreak of the disease often follows the breaking up of a virgin soil.

3. SEASONAL RELATIONS.—It is a disease of the warmer months, a tolerably high temperature being necessary for its development. A mild type occurs in the spring and early summer, and a severe type in the late summer and autumn months.

4. AGE, SEX AND RACE RELATIONS.—It attacks all ages, but most frequently children and young adults; the

male sex mostly because of the greater exposure of man to the virus in virtue of his occupations; and the white races chiefly, for the negro and native races, though not immune, are much less rarely attacked. It has recently been shown that while the adult natives in a malarious region may be free from the disease, an enormously large percentage of the young children suffer to a greater or less extent, though always less than new arrivals. The malarial parasite to be presently described, though absent from the blood of the adults, is present in the blood in a vast proportion of the children, and though it may cause but little trouble in them, it is capable of causing dangerous ague in new arrivals. It would thus appear that native children possess a certain degree of congenital immunity, and that adult natives acquire a further degree of immunity.

5. THE PLASMODIUM MALARIÆ.—This organism is the real cause of malaria; without it the influences above mentioned are generally believed to be ineffectual. It is a protozoon, and was first discovered by Laveran in 1880, and so faithful is his description that very little of it has been altered up to the present day. It resides in the red blood-cells, being at first a small, clear hyaline amœboid mass of protoplasm. It soon acquires a black pigment, formed at the expense of the hæmoglobin, which, as the parasite grows in size, gathers from the periphery towards the centre of its protoplasm. When fully grown it loses its amœboid movement and assumes a somewhat rounded, though never quite round form, and occupies when largest, as in the simple tertian variety, almost the whole of the red blood-cell, which, often somewhat enlarged, is seen as a thin pale rim around it. It is during this growth of the parasite within the red blood-cell that the intervals between the patient's feverish attacks occur. The protoplasm of the parasite now breaks up into a number of spheres or ovoid bodies, so arranged in a

radiating manner outwards from the central pigment mass as to give the appearance of a rosette. These sphere-shaped bodies rupture the red blood-cell and escape into the blood-plasma. Coincidentally the temperature rises and the patient suffers from fever, probably from the liberation of some pyrogenetic substance into the blood fluid at the same time as the spherules escape. These spherules are spores, and after being for a time free they are believed to fasten themselves to other healthy red blood-cells, which they enter in turn, and growing therein produce the same cycle as before. No other change is ever seen in the circulating blood, and this sporulation or asexual reproduction may go on indefinitely, or last for about two weeks only, recovering spontaneously or after treatment; but recurrence is very frequent. In the quotidian variety (if a true quotidian really exists, which is doubtful), in which the fever returns daily, the life-cycle is completed in twenty-four hours. In the tertian variety, in which the fever returns every second day, the life-cycle is completed within forty-eight hours. In the quartan variety, in which the fever returns every third day, the life-cycle is completed within seventy-two hours.

The disease may be arranged in two great clinical groups according to its severity, viz., benign and malignant. The former, the benign group, comprises the winter-spring parasites, viz., those causing quartan and benign tertian ague; while the latter, the malignant group, comprises one form only, the summer-autumn form, which causes malignant quotidian or tertian or irregular ague.

Each variety has a special form of parasite. It is largest in the benign tertian type, where it is actively amoeboid and has many fine pigment granules. When it is fully grown it is about the size of a red blood-corpuscle, and hence the red blood-cell which contains it is swollen and decolorised. In sporulation, which occurs immediately before the end of the cycle, the segments number usually

from fifteen to twenty. In the quartan variety, the parasite does not usually grow quite so large, its amœboid movements are slower, and its pigment granules are coarser and darker. In sporulation, which begins several hours before the end of the cycle, the segments number only six to twelve, but are generally arranged in beautiful rosettes. The parasites of these two varieties exist in the blood in great groups, all the members of which are approximately at the same stage of development; but two separate groups of tertian parasites, reaching maturity on alternate days, may enter the same body, and hence cause quotidian paroxysms. This double tertian infection is more common than the entrance of only one group. Infection with multiple tertian groups is rare. Again, two separate groups of quartan parasites may be present, reaching maturity and causing paroxysms on two successive days, with a day of intermission following—*i.e.*, the double quartan type. If three separate groups of quartan parasites are present, the paroxysms occur daily—*i.e.*, are quotidian. The benign quotidian variety may thus arise from either double tertian or triple quartan infection. The quotidian form of ague may also arise from a special type of parasite, which belongs to the summer-autumnal group and is the common tropical variety of malarial fever. Its life-cycle is more irregular and may be completed in twenty-four hours or longer. At first all the parasites appear to be at the same stage of development, but later forms at different stages appear. They do not mature in groups. It is a small parasite, rarely exceeding one-third to a half the size of a red blood-cell. The pigment is scanty. It often assumes a quiescent ring form. The earlier stages of development only are seen in the peripheral circulation, the later are confined to the blood of certain internal organs, such as the brain and spleen.

It is here that sporulation occurs, the spores, being very minute and usually six to eight in number, not forming a

definite rosette. There is probably only one variety of malignant parasite having a life-cycle varying considerably (from twenty-four to forty-nine hours), thus giving rise to the belief formerly held that a separate tertian and perhaps other varieties existed.

These life-cycles explain the remarkable periodicity shown by the different varieties, the attack of fever being always coincident with sporulation and disappearing during the interval required for the growth of the spore within the red blood-cells, until it reaches maturity, when sporulation and another paroxysm will occur. It is thus seen that there may be great variety in the type of ague present in any case, as it will depend upon the kind of infection, whether simple or mixed. It is also possible that all the varieties of the parasite have not yet been discovered. We have next to explain how the disease is infectious and passes from the sick to the sound. Experimentally it has been shown that if some of the blood of a sick person, taken during a paroxysm, be injected into a vein of a healthy individual, the latter acquires exactly the same type of ague as the first suffers from; but how does such transference take place in nature? Manson explains it by his mosquito theory, which, by his own labours and those of MacCallum, Ross and many others, has now had practically every link in the chain of proof completed. In the blood of patients affected with the summer and autumnal fever, crescent-shaped parasites, with, generally, central clumps of pigment, are seen. If the blood be withdrawn and watched under the warm stage of a microscope these crescents are seen, in about twenty minutes, to assume a globular form and then to throw out a number of flagella which show active movement. Certain fully-grown benign tertian and quartan parasites, which resemble the sporulating forms but do not themselves sporulate, may form similar flagellate bodies without first becoming crescents, which are never seen in these varieties.

This exflagellation takes place only after the blood is withdrawn, never so long as it remains within the body of the patient. The mosquito (genus *anopheles*) provides the necessary link. It bites the patient and the blood passes into its stomach, where exflagellation then takes place. The sphere-shaped bodies which give out these flagella have a faintly-staining protoplasm, and can thus be distinguished from other and larger sphere-shaped bodies with a coarsely-granular pigmented protoplasm staining somewhat deeply. They are present together, and presently one of the flagella becomes detached, and seeking out one of the granular spheres enters it and so impregnates it; the flagellum being regarded as the male element and the granular sphere as the female cell. Only one flagellum enters an individual cell. After impregnation the granular sphere becomes pear-shaped or beaked, and the pigment having collected at the broader, posterior end, it darts about point first. It is now named a travelling vermicle. The vermicle passes through the wall of the mosquito's stomach to its peritoneal surface, and, becoming round and encapsulated, grows in size until it forms a wart-like swelling visible to the naked eye upon the outer wall of the mosquito's stomach. These wart-like growths are multiple. Their contents soon show a number of minute non-pigmented spherical bodies, which give rise in turn to spindle-shaped filaments or embryos, which finally rupture the capsule and are carried by the blood or lymph to the salivary gland. This generally occurs in from one to two weeks after the mosquito bite and blood ingestion. When the mosquito now bites a fresh individual, it injects, along with the venom, some of these embryos, which in time penetrate the red blood-cells and develop into fully-formed malarial parasites, the clinical symptoms of *ague* generally appearing after a period of about ten to twelve days' incubation. This explains the whole life-history of the malarial parasite, and it is impossible any longer to doubt that infection by the mosquito

is one at least of the ways by which malaria spreads. That it is the only way is not yet indubitably proved, but the recent experiments of Sambon and Low, authorised by the British Colonial Office at the instigation of Manson, go far to do so. Those gentlemen have demonstrated that it is possible to live throughout the malarial season in one of the worst haunts of that scourge (the Roman Campagna) without contracting the disease, by simply taking precautions against mosquito bites. They went about freely during the day, when the mosquitoes of that region rarely bite, and retired at sunset to a specially constructed mosquito-proof hut. Professor Grassi has corroborated this observation on a much larger scale, in which 104 persons were similarly protected, and he believes that it is now conclusively proved that malaria can be transmitted only by the bites of mosquitoes belonging to the genus *anopheles*, and that practical protection therefrom is already a feasible procedure. It is possible that different species of *anopheles* may convey different species of the malarial organism, and it is also asserted that occasionally other species than the *anopheles* may convey the parasite.

Morbid Anatomy.—1. *The simple intermittent forms* (benign tertian and quartan).—These are rarely fatal, and hence we do not know much about the tissue changes which occur. The red blood-cells show the parasites. There is anæmia, due to destruction of the red cells, and there is pigment present in the blood and also deposited in the viscera, due to the transformation of the hæmoglobin. The spleen is usually enlarged, sometimes greatly.

2. *The remittent and pernicious forms.*—The blood shows similar changes. The anæmia is more marked. Poikilocytosis is common. The spleen is enlarged and sometimes greatly pigmented. The liver is enlarged and pigmented, presenting a dark grey colour. The capillaries

show many phagocytes and parasites. Cirrhosis and areas of disseminated necrosis may be present. The kidneys show less marked pigmentation. There is parenchymatous degeneration, more or less severe, and sometimes even necrosis of the epithelium of the convoluted tubules and hæmorrhages. The brain usually shows a reddish discoloration, and the vessels may show many parasites, with partial or complete destruction of the red blood-cells.

Symptoms.— They vary somewhat according to the clinical type of fever which is present. It is convenient to speak of Intermittent, Remittent, Irregular, Pernicious and Long Interval Fevers.

1. The Intermittent Fevers are caused by the benign tertian and quartan parasites, and hence occur daily, or every second or every third day, giving the quotidian (which is really a double tertian or triple quartan), the tertian and the quartan fevers. They all show the three stages—the cold, the hot and the sweating stage—which follow each other in regular sequence, and are so characteristic of an attack of ague. In the cold stage there is chilliness, which develops into a rigor with its characteristic shivering and cold external temperature. The rigor may be preceded by frontal headache, lassitude, yawning and uncomfortable sensations. This stage lasts from four or five minutes to an hour or more. In the hot stage the skin becomes hot and burning to the touch, the face is flushed, and there is a throbbing headache. The tongue is dry and there is great thirst. The pulse is full and bounding, and the surface temperature (axilla) high (often 105 to 106°F.). The duration of this stage varies from one to two or more hours. It is generally in an inverse ratio to the cold stage—the shorter the cold the longer the hot, and *vice versa*. In the sweating stage the symptoms of the hot stage diminish and there is free perspiration, the patient feeling a general relief. This lasts from two to three hours, by which time the attack is over.

2. Remittent Fever.—It begins with a rigor or other feverish symptom and is followed by a rise of temperature. This continues for a time, remitting at intervals, especially if attempts at sweating occur. The remissions in the fever may be well marked, but the temperature never falls to normal. At other times they are very slight and the fever is characterised as being continuous, or continued fever. When vomiting is prominent it is termed “bilious remittent”; when it resembles typhoid it is called typho-remittent or typho-malarial fever. The æstivo-autumnal or malignant parasite is found in the remittent fevers, the crescent-shaped forms being found after the fever has lasted for over a week. The same malignant parasite is found in the irregular and the pernicious types of fever, the latter signifying that one organ or group of organs bears the brunt of the attack. The long interval fevers occur in those who have had no attack for months or years, indicating a “latent phase” of the parasite and that it is able to become active again only under exceptionally toward vital conditions on the part of its host.

Treatment.—The treatment of intermittent fever is divided into two parts—palliative, during the paroxysms, and curative, during both paroxysms and intermissions.

During the cold stage the patient should be put to bed, and every attempt made to restore warmth to the body; hot tea or coffee may be given, and friction applied to the extremities. A hypodermic injection of morphia is most comforting to the patient during this stage. Relief during the hot stage is obtained by sponging the body with cold or tepid water. Ice or iced drinks are indicated to relieve the thirst. Careful protection from cold draughts, and wiping the perspiration from the body with warm flannels, will promote the comfort of the patient during the sweating stage.

The curative measures consist in the administration of a brisk cathartic, followed by the exhibition of large

doses of quinine. The most successful mode of procedure appears to be as follows:—Ten or twenty grains of the sulphate of quinine should be given in solution during the sweating stage, followed by one or two grain doses three times during the day. Quinine is the chief remedy for ague, and it may be given by mouth, hypodermically, or injected into a vein. If taken in five grain doses thrice daily, once a week, or even a small dose daily or every second or third day, malaria is often averted. During the attack a big dose of ten to twenty or more grains of quinine should be taken, and repeated if necessary. Either the sulphate or hydrochlorate may be used. Hypodermically, it is well to employ the acid hydrochlorate and to give in the first instance about seven grains in one c.c. of distilled water. Twice or three times that dose may be necessary. It should be injected deeply into the muscles, and the most rigid antiseptic precautions must be taken, so that the syringe may be absolutely clean. It is only desirable to use hypodermic or intravenous injections in very severe cases, where life is in danger, or where in the attack the nausea absolutely prevents all medicine being taken by the mouth. Various remedies are required to retard the cinchonism which is so troublesome, and of these hydrobromic acid is probably the best. Quinine has a quite definite toxic action on the plasmodia, and not merely arrests or retards sporulation but causes a peculiar shrinking or shrivelling of the organism. No other drug yet discovered has a similar effect. Arsenic is certainly often useful, and iron greatly benefits the anæmia or cachexia so commonly associated with malaria. The spleen may give trouble when enlarged, and mercurial inunction or painting the skin over the splenic region with iodine has been found serviceable in bringing about reduction in size of that organ. Great care should be taken to avoid exposure to fresh attacks, and wet or cold, even in a non-malarial land, are apt to induce an attack. The prophylaxis of malaria is most

interesting. Once the pools of stagnant water and the marshy ground which form such ideal breeding preserves for mosquitoes are done away with, the risks of so-called malarial countries become greatly minimised. Filling up the pools, or, if that is for some reason impossible, pouring a thin layer of kerosine oil on the surface, effectually kills off all mosquitoes. The work being carried out at present in West Africa and elsewhere upon these lines has ceased to be experimental and has become an established prophylactic procedure.

HÆMOGLOBINURIC FEVER.

Syn. *Blackwater Fever, West African Fever.*

DEFINITION.—A specific fever characterised by hæmoglobinuria, jaundice, and vomiting.

This disease is particularly prevalent in tropical West Africa, but it is also met with in Central and East Africa, in Cuba, and in tropical parts elsewhere

Etiology.—Its true causation is still a matter of doubt. Three views are held regarding it:—

1. That it is malarial. In favour of this view is the fact that malarial parasites are found in about half of the cases, and even when they cannot be demonstrated the condition may be one of "intoxication of malarial origin" (Crosse). Against it is the absence of the malarial parasite in nearly half of the cases, the frequent absence of true relapses so common in malaria, and the probability of a mixed infection being present, as malaria is common in many blackwater fever districts.

2. That it is caused by quinine. The wide geographical distribution of malaria, in which the administration of quinine is so universal, and the restricted geographical

distribution of blackwater fever makes this view very improbable.

3. That it is caused by a specific micro-organism allied to the malarial parasite. Dr Sambon holds that it may be similar to the hæmatozoon which causes hæmoglobinuric fever in cattle, and that paroxysmal hæmoglobinuria is merely a mild form of hæmoglobinuric fever.

Morbid Anatomy.—There is usually marked anæmia, there being diminution in number and irregularity in shape of the red blood-cells, and also diminution (both relative and absolute) in the quantity of hæmoglobin. There is also well-marked leucocytosis. There may be much pigmentation in various organs, but this may be due to an antecedent or coincident malarial infection.

Symptoms.—It usually appears in the third, rarely in the first year of residence in a blackwater fever district. The onset is generally sudden, with or without foregoing fever, commencing with a rigor, headache, pain in the epigastrium and loins, and severe bilious vomiting. The temperature rises to 103 or 104°F. and the pulse is small and quick, not full and strong as in intermittent fevers. There is much restlessness and anxiety. The urine, at first reddish, soon becomes dark brown and thickish, like porter; and when it is allowed to stand the sediment shows large granular hæmoglobin and hyaline casts, much granular pigment and detritus, a few red blood-cells, and epithelium. Micturition usually becomes frequent. Jaundice appears after a few hours. These symptoms usually abate in about ten to twelve hours in favourable cases, the temperature falling rapidly and a profuse and exhausting perspiration setting in. There may be no recurrence, or more usually another attack next day, sometimes followed on subsequent days by repeated recurrences. In unfavourable cases the temperature keeps

up, the vomiting persists, the urine becomes suppressed, the brain affected, and usually uræmia precedes death.

Diagnosis.—(1) From yellow fever it is distinguished by the early appearance of the dark coloured urine, the absence of blood from the vomit, and its more usually rapid course. (2) From paroxysmal hæmoglobinuria by the greater severity of its symptoms. (3) From toxic hæmoglobinuria, such as follows the administration of potassium chlorate, sulphuric, hydrochloric, pyrogallie and carbolic acids, arseniuretted hydrogen, naphthol, carbonic oxide, anilin, &c., by cold and exertion, and by the history of the case.

Prognosis.—Recovery is common after single or repeated attacks, but when the symptoms are severe and the fever does not abate, a grave result should be feared. The mortality is often as high as sixty per cent. of those attacked.

Treatment.—It is probably wise to give quinine, and to give it in full doses, and to disregard the statements that quinine, even in small doses, is harmful in this disease. Calomel in large doses is much used in West Africa, and some so-called astringents, especially tannin in 15 grain doses thrice daily, have a great reputation. Perhaps the most important advice to a convalescent patient is to avoid cold weather, as it may bring on a fatal relapse.

PSOROSPERMIASIS.

This includes a number of different but allied conditions produced by parasites belonging to the simplest class of the animal kingdom—the protozoa. Apart from malaria and amœbic dysentery, the commonest pathogenic member of the group is the *coccidium oviforme* of the rabbit, which

(almost always if not universally present in certain districts in the alimentary canal of rabbits) causes lesions in their livers, in the form of whitish nodules of varying size. Microscopic examination shows that they are due to the coccidium invading the walls of the bile ducts. It has an oval capsule surrounding the protoplasm, which stains readily, the nucleus remaining pale. It is uncertain if the intestinal and hepatic forms of the parasite are the same or different. It does not seem, except occasionally, to give rise to serious changes affecting the animal's health. Other coccidia have been recorded to cause intestinal mischief in pheasants, lambs and cattle. Another form, the *sarcocystis miescheri*, also ovoid but containing a number of sickle-shaped bodies, is fairly frequent within the sarcolemma of the muscles of the pig. Outbreaks of similar parasites have occurred in man, which may be grouped as internal and external.

1. INTERNAL PSOROSPERMIASIS.—The liver has shown in most cases similar white nodules to those seen in rabbits. Similar nodules resembling tubercles have been found in the spleen, kidneys, ureters, peritoneum, &c. The true nature of these cases was recognised because they proved fatal. It is quite possible that other cases recover, but no proof of this has yet been brought forward, and similar lesions have not been found postmortem in cases dying from other causes.

2. CUTANEOUS PSOROSPERMIASIS.—Several cases of extensive skin disease (face, body and limbs), due to coccidia-like bodies have been recorded. These bodies were found in great numbers in the pus. A generalised disease, like tubercle, was found after death, the coccidia being found in all the lesions. Paget's disease of the nipple has been described as showing protozoal-like bodies intermediate between these coccidia and the so-called cancer bodies, but the exact nature of all these bodies found in Paget's disease, in malignant tumors, and in molluscum contagiosum, is still in dispute.

Treatment.—Little can be said about the treatment of this condition excepting that symptoms should be combated as they occur.

SLEEPING SICKNESS.

Etiology.—1. THE GEOGRAPHICAL DISTRIBUTION.—This disease has long been prevalent in the western coast line of Africa and its hinterland, from Senegal along the Gulf of Guinea to Portuguese West Africa, and extending up the Congo as far as the Stanley Falls. Of late years (within the last three or four) it has extended to East Equatorial Africa, mainly centring round Lake Victoria Nyanza and its waters for a radius of some 10 miles from its shores. It was this recent extension which stimulated investigation into its nature. So far as East Africa is concerned it appears to be chiefly contracted in this region, but it is also prevalent in its hinterland, particularly to the east and south-east. It is thought, however, that it is not contracted in this latter region.

2. THE PEOPLE.—The poorer classes, particularly among the agriculturists, are chiefly attacked. The chiefs and better-class natives suffer much less, and so far only one or two cases have occurred in Europeans.

3. THE GERM.—It has been conclusively proved that a trypanosoma is the true cause of the disease, though the Portuguese authorities seem still disinclined entirely to give up the claims of a minute diplococcus. It is an actively motile twisted body seen in the blood in all stages, and in the cerebro-spinal fluid mainly in the later stages of the disease. Its length varies from 16 μ to 24 μ and the width at its greatest part is about 2 μ . It possesses, like all other trypanosomata, a protoplasmic body, which is blunt at its posterior end and pointed at its anterior end, the latter possessing a long whip-like flagellum. Unlike all other forms the posterior end goes first when it moves. It stains well

with Leishman's modification of Romanowsky's stain. The nucleus stains red and is generally large and situated in the posterior end. The protoplasm stains unevenly and of a pale blue colour. It usually shows some chromatic granules. There is a small body which stains a deep red (far deeper than the nucleus) situated near the posterior end, posterior to and generally outside an oval clear vacuole. The nature of this small body is in doubt. It is held to be a micro-nucleus by Plimmer and Bradford, while Laveran calls it a centrosome. There is a clear unstained undulating membrane running along one side of the organism. The external edge of this membrane appears to take origin from the micro-nucleus at the posterior end, and at the anterior end to become free and form a long whip-like process—the flagellum—which is usually longer than in other trypanosomes.

4. METHOD OF ENTRANCE into the patient.—It enters the blood through the bite of a fly, the *Glossina palpalis*, which, like *Glossina morsitans*, the carrier of the trypanosome which causes the disease known as nagana to cattle, is a species of the genus *Glossina*. The fly in biting a patient withdraws some of the trypanosomes with the blood, and subsequently in biting a sound person injects them into the latter's blood. It therefore plays the same part as the mosquito does in malaria, except that the trypanosome of sleeping sickness, unlike the plasmodium of malaria, does not appear to be able to live more than a short time within the body of the fly, and there is, further, no evidence at present that it undergoes any development therein. It multiplies within the blood, fission being so far the only known method of reproduction. It was at first thought to be restricted to the blood for an indefinite period, which is sometimes very prolonged, and ultimately to enter the cerebro-spinal fluid, when shortly thereafter the definite symptoms of sleeping sickness begin. This relationship is now regarded as doubtful, further research

seeming to show that the commencement of fever or other symptoms is in no way co-related to the entrance of the trypanosomes into the cerebro-spinal fluid, that in many cases they never find their way there at all, that they may come and go from it as from the blood, but that when present in it in large numbers, which is rare, there is usually an access of fever. The disease is transmissible to monkeys, in whom it runs a more rapid course than in man. Dogs and cats are partially susceptible.

Morbid Anatomy.—The chief changes are seen in the nervous system in the form of a chronic meningitis of the brain and cord and inflammation of the underlying nervous tissue. The macroscopic changes are slight but the microscope shows a uninuclear leucocyte infiltration on the surface of the meninges, invading the sulci, and also the perivascular spaces around the blood-vessels in the superficial areas of the cerebrum, pons, medulla, and spinal cord. Enlarged glands are seen in all cases, and are believed to be a feature of the disease. The trypanosome is found most frequently in the blood, sometimes in the serous cavities, and in the cerebro-spinal fluid, which may also contain uninucleated leucocytes. The red blood-cells are generally diminished, and there is a relative increase of the large uninuclear leucocytes.

Symptoms.—They begin insidiously and are at first indefinite. A general lack of energy, headache and other transient pains—*e.g.*, in the chest—may be complained of. The aspect usually becomes dull, there is a marked delay in answering questions, and a fine tremor appears in the tongue and hands. Fever is an early symptom. There is a rise of temperature to 101°F. or 102°F. in the evening, followed by a morning fall to normal or below it, sometimes amounting to 4° . This continues for three or four weeks, when the temperature becomes irregular

and, for one or two weeks preceding death, usually subnormal. In very acute cases it may be subnormal throughout. The pulse is always quick (90 to 130), of very low tension, and only towards the end does it become infrequent. Respiration is increased. The appetite usually remains good, and constipation is a constant feature. The skin may become rough, especially in chronic cases, and show papulo-pustular eruptions. The lymphatic glands are enlarged. Drowsiness and lethargy appear if not previously noticed and soon become more pronounced; but it is not a true or deep or continuous sleep, as the slightest touch will cause the patient to rouse up. Fine tremors are now marked in the tongue and arms. They are a marked feature of the second stage of the disease. *Emaciation and general weakness*, which have gradually come on, become more marked, so that difficulty in walking is followed by inability to walk at all and the patient becomes bedridden (the third stage). The knee jerks, at first exaggerated, diminish; loss of control of the sphincters follows. Epileptic fits sometimes occur. Flexor contractions at the knees and hips are common, the drowsiness increases and passes on to coma and death.

Diagnosis.—The early well marked fever with evening rise and morning fall, the low tension and quick rate of the pulse and the fine tremor are the most important symptoms. The blood should be examined at intervals for trypanosomes, and also the cerebro-spinal fluid obtained by lumbar puncture.

Prognosis.—The cases are believed always to end fatally. The average length of the illness is four to eight months, it rarely lasts over a year, and is not uncommonly very short. In most cases death is due to the occurrence of some complication caused by a bacterial infection, *e.g.*, pneumonia, purulent meningitis, pleurisy, &c.

Treatment.—The treatment of this disease has been most unsatisfactory: purgatives, quinine, and, best of all, arsenic, are the remedies generally recommended; but benefit has been merely temporary except in isolated cases. The total destruction of the fly which carries the parasite, it is needless to say, would be the best possible method of stamping out the disease.

II.—DISEASES CAUSED BY WORMS.

THE TREMATODES—DISTOMIASIS.

The members of this group, generally called the fluke worms, have flat leaf-like bodies provided with one or two suckers, by which they cling. They have a mouth and branched alimentary canal, but no anus or body cavity. They are all hermaphrodite except *Bilharzia hæmatobia*; and certain stages of their somewhat complicated development must be passed within the body of an intermediate host, such as the pond snail. Several members of the group may occur as human parasites, mostly within the bile ducts, liver or duodenum, but *Distoma Ringeri* invades the lungs, causing frequent hæmoptysis. While common in Japan and China they are rare in this country. The symptoms are generally somewhat indefinite. There may be anæmia and intestinal irritation, with escape of the ova or the worms per anum. There may be jaundice and ascites, suggesting hepatic cirrhosis. *Bilharzia hæmatobia* is an exceptional member of the group in several respects. The sexes are in separate individuals, the male being about half-an-inch long and the female more. The latter is narrow and is carried during copulation in a deep groove (the “gynæcophoric canal”) on the ventral surface of the male. The adult worm inhabits the blood-vessels, the portal vein, the mesenteric vein, and the vessels of the bladder and rectum,

and does little or no harm. It is common in the Cape and Egypt. The ova, on the other hand, give rise to hæmaturia and diarrhoea. They are oval or pear-shaped and have a hard transparent shell armed with a sharp spine, which is generally situated terminally in bladder cases, and laterally in rectal cases. The ova pass from the blood-vessels into the submucosa, mucosa and cavity of the bladder or rectum, causing in the one case hæmaturia, with or without painful micturition, calculus, and ultimately cystitis, and in the other case catarrh and papillomatous projections of the mucous membrane, with resulting diarrhoea, tenesmus and wasting. The vesiculæ seminalis and kidneys may be involved later. The ova are easily recognised in the urine, and many of them contain fully-formed ciliated larvæ, which may be seen to burst the egg cell longitudinally and become free. The stages in the development are unknown. There may be an intermediate host, and they may enter the human body in water in the form of an encysted cercaria. Direct infection from the embryos entering the anus or urethra or penetrating the skin during bathing is believed to occur.

Treatment.—*Bilharzia hæmatobia* should be treated by administering turpentine in capsules (10 to 30 minims), and if cystitis be present, which is probable, other suitable measures may be tried. It is extremely difficult to cure the condition. *Distomiasis* can be treated with thymol where the flukes in question are in the intestine. Other forms are generally beyond reach of any radical measures for their destruction.

THE CESTODES—TAPE-WORMS—HYDATIDS.

Tænia solium, *Tænia mediocanellata* and *Bothriocephalus latus* are the common forms found in man. Others, e.g., *Tænia tenella*, *Tænia elliptica*, &c., are so rare that they do

not require special notice. A detailed description of the three common forms cannot be given here, and the reader unfamiliar with them will find them in any text-book upon Zoology. A short summary of the main facts will suffice. Each worm possesses a small head about the size of a small pin head or smaller, a short thin neck, and a long body measuring many feet. There is no mouth or alimentary system or body cavity. It attaches itself to the mucous membrane by its head, usually called the "scolex," which may possess both hooklets and suckers for this purpose. *Tænia solium* has both, *Tænia mediocanellata* has four suckers only, and *Bothriocephalus latus* has two lateral slit-like suckers. The body, usually called the strobila, lies free within the cavity of the intestine. It is flat and segmented, the segments, usually called proglottides, are small next the head but gradually increase in size thereafter, until towards the end of the body they measure over half-an-inch long, and about a quarter of an inch broad in either *Tænia solium* or *Tænia mediocanellata*, but are broader than long in *Bothriocephalus latus*. They are produced by budding from the head, and each contains when mature both male and female organs. The testes and ovaries exist only for a short time, so that each mature segment contains chiefly a branched uterus and eggs. There is a genital pore, situated laterally in *Tænia solium* and *Tænia mediocanellata* but ventrally in *Bothriocephalus latus*. The fertilised eggs may leave the segments by the genital pore, or a whole segment or number of segments may become detached, as they are loosely attached to one another at the posterior end, and escape with the fæces. The eggs, or even a whole proglottis, which may contain as many as 50,000 eggs, may be swallowed by an animal with its food. The egg shell is broken in mastication or dissolved by the gastric juice, and the embryo, a small body provided with six hooklets, escapes and bores its way through the wall of the alimentary canal and is carried to the voluntary

muscles or other situation, where it becomes encysted. It increases in size, forming a cyst filled with fluid. This is known as the prosclex stage. The head now appears by a process of invagination of one part of the wall of the cyst, and in the inner side of this the hooklets or suckers, or both, are formed. These are everted when the head is fully formed and the remaining part swells into a bladder. This is the bladder-worm or cysticercus stage. In the case of *Tænia solium* this stage is usually seen in the muscles of the pig (the cysticercus cellulossæ constituting "measly pork"), and is on an average about one-sixth inch in diameter. In the case of *Tænia mediocanellata* it is rather smaller, and is seen in the muscles of the ox; while in *Bothriocephalus latus* it inhabits the pike and other fresh-water fish. The cysticercus is surrounded by a true capsule of its own formation. Outside this is a fibrous tissue capsule, the result of chronic inflammation in the surrounding tissues. When the flesh of any of the animals containing cysticerci is eaten by man, the capsules are dissolved, the head escapes and the bladder disappears. When the head reaches the intestine it attaches itself and begins to bud off segments. The *Tænia mediocanellata* is the commonest in this country, America, &c., where beef is the staple animal food, but *Tænia solium* is commonest in Germany, where pork is much eaten, and *Bothriocephalus latus* in Switzerland, where dried raw fish is indulged in. Man is therefore the adult host, while the pig, ox or fish is the intermediate host. In rare cases man plays the part of the intermediate host, the cysticercus cellulossæ being found in the heart, brain, subcutaneous tissues, &c.

Symptoms.—The symptoms caused by tape-worms are very indefinite. They are often entirely absent, and the presence of the worm becomes known only by the escape of segments per anum. The Abyssinians are said to court the presence of one or more tape-worms, under the belief

that their general well-being is thereby increased. When symptoms are present they are referable either to intestinal irritation (pain, nausea, vomiting, slight diarrhoea alternating with constipation), to constitutional effects (wasting anæmia, particularly in the case of *Bothriocephalus latus*, debility), or to reflex irritation (giddiness, twitching of muscles, or even convulsions).

Treatment.—Begin treatment by clearing out the bowels and then starve the patient before administering the anthelmintic. It is well to give the patient the dose in the early morning on an absolutely empty stomach, and either to give in one dose one to two drachms of the extract of *felix mas* made up as an emulsion with cinnamon water or else to divide into two or three doses given every half hour. Follow with a dose of castor oil. Pomegranate root, pumpkin seeds, and many other anthelmintics may be substituted. Usually a second or third dose is necessary, and the stools should be carefully examined so as to make certain that the head of the worm has been got rid of. Remember there may be several worms present and the life-history of each individual *tænia* may, if not removed, be a matter of years.

TÆNIA ECHINOCOCCUS—HYDATIDS.

This tape-worm, the most important of the group so far as man is concerned, is in its adult stage very small (about quarter of an inch), but it makes up for this by the size of its scolex stage, which is often very large. Further, it is the scolex stage which inhabits man, who is thus the intermediate and not the adult host as in other tape-worms. The adult worm inhabits the intestine of the dog and wolf, and echinococcal disease in man is consequently common in countries where men and dogs live in close association,

such as Iceland and Australia, though it is met with in varying frequency in all places. Infection is usually through the food: often by means of imperfectly cleansed and cooked vegetables. The adult worm has a head, with a diameter of about $\frac{1}{40}$ inch, and three segments, of which the last only is sexually mature. The head possesses a rostellum, a double circlet of hooklets, and four suckers. The embryos when liberated from the egg bore their way through the intestinal wall and are carried by the blood to some one or more of the organs, where they become encysted, forming the scolex stage, as in other tape-worms. This is known as the hydatid. Both the true and false capsules are of considerable thickness, and they usually increase greatly in size as the hydatid grows. The true capsule consists of two parts—an outer or ectocyst (a thick laminated highly refractile coat composed of chitin) and an inner or endocyst (a thin parenchymatous layer which is the vital part of the parasite). The contained fluid is colourless and transparent and contains a large quantity of common salt but no albumen. The disease would not be so serious if the development stopped here. Usually it does not do so but forms secondary or daughter cysts by budding from the endocyst, often so many of them that they completely fill the cavity of the mother cyst. The cyst layers—ectocyst and endocyst—are reversed in position, the endocyst being the outer, and from it numerous heads with hooklets and suckers may develop. These secondary cysts are called brood capsules. They are sometimes still further complicated by cysts (granddaughter) being in turn formed within them. Heads are always developed from the endocysts, and may form in all the cysts, but more usually some of these remain barren. The secondary cysts may develop either exogenously or endogenously in relation to the primary cyst. The latter is the more common, giving the external appearance of one cyst, while the former shows several or many cysts joined together

at their sites of origin. These may subsequently be joined by fusion and compression into what is apparently one mass—"the multilocular hydatid." Several or many quite separate hydatids may be present. The liver is the commonest seat, the peritoneum coming next in frequency, but almost every organ in the body may be invaded.

Symptoms.—They are due to the mechanical pressure exerted by the growing hydatids, and are usually absent until it grows to a considerable size, sometimes even throughout life. The pressure results depend upon their anatomical situation and size; thus they will become manifest when situated in the brain sooner than when situated in the liver or peritoneum. It is noticeable that pain is rarely complained of. When palpable externally a peculiar vibration, known as the "hydatid thrill," may be felt on percussion over the cyst; but it is not peculiar to hydatids, as it may be felt over other similarly situated cysts.

Diagnosis.—The history will often make a diagnosis from simple or malignant tumor or gumma, &c., fairly probable, but an absolute decision can only be come to by the discovery of hooklets or scolices in the fluid, which may be withdrawn from the tumor by a fine hypodermic needle. This must be done with care, and signs of collapse carefully watched for during the procedure.

Treatment.—The treatment for a hydatid cyst depends much on its position. Frequently tapping is sufficient to effect a cure so far as that cyst is concerned, but free evacuation of the cyst is common practice. Fortunately the presence of this *tænia* in dogs is exceptional in this country; but care should be taken to prevent even the possibility of infection.

THE NEMATODES—ROUND AND THREAD WORMS.

These worms are all elongated and cylindrical, although their length may be very short. The body wall is unsegmented and provided with a strong chitinous external cuticle. There is a mouth and a complete alimentary canal ending in an anus towards the posterior end of the body, and lying within a distinct body cavity. The sexes are in separate individuals. The genital organs, consisting of coiled thread-like tubes, open by a distinct genital pore, usually situated posteriorly in the male and anteriorly in the female. Development is direct in most cases, no intermediate host being required. The commonest forms in this country are *Ascaris lumbricoides* and *Ascaris* or *Oxyuris vermicularis*. They inhabit the intestine of man. Other important forms which also live in the intestine are—*Trichocephalus dispar*, *Anchylostoma duodenale* and *Trichinæ spiralis*. The other chief members, viz., *Dracunculus medinensis* and the filariæ, do not inhabit the intestine.

ASCARIS LUMBRICOIDES—THE ROUND WORM —ASCARIASIS.

The female is about ten to fourteen inches, the male about four to six inches long. The worm is of a pale yellowish-brown colour and tapers at both ends, the anterior end being perforated by the mouth, which has three small lobes, while the anus lies just in front of the posterior end. The genital aperture in the male opens into the rectum close to the anus, while in the female it opens on the ventral surface at the junction of the anterior and middle thirds of the body. The ova are fertilised in the uterus, and are discharged in large numbers from each worm. They are oval, measure about $\frac{1}{340}$ inch in length and $\frac{1}{440}$ inch in

width, and are discharged with the *fæces* of the human host. They enter by the drinking water and develop into the adult worm in the small intestine. One is the usual number, but two or many may be present in the same host. They are also given to wandering, and may migrate downwards into the colon or rectum, or more rarely into the bile passages (sometimes causing jaundice), stomach (sometimes vomited), *œsophagus*, larynx (causing asphyxia), lung (gangrene), nose and outside, especially during sleep, being found on the pillow, &c. When many are present they cause obstruction of the bowel. Rarely they penetrate the bowel, causing peritonitis.

Symptoms.—When present they point to intestinal irritation and reflexly excited nervous disturbances. They are most marked in children. Those chiefly noticed are disturbed sleep, grinding of the teeth, itching at the nose, irritability, muscular twitchings, or even convulsions.

Treatment.—Quite the best remedy is *santonin*, one to six grains, according to age. Preliminary starvation for twelve hours, next a dose of castor oil, followed by the *santonin*, and then a second dose of castor oil will be found an excellent plan for speedy clearance. Not infrequently some transient giddiness follows the use of *santonin* but it soon passes off.

OXYURIS VERMICULARIS—THE THREAD-WORM.

The female is about one-third inch, the male one-sixth inch long. The colour is white. The mouth, situated anteriorly, has three small lobes. The posterior end is tapering in the female and blunt in the male. The ova are fertilised in the uterus and escape in great numbers

with the fæces, as do also the worms themselves. The ova are oval and about $\frac{1}{500}$ inch long and $\frac{1}{1000}$ inch broad. They gain entrance to another individual or to the same individual through uncleanly habits, or by the drinking water or food, and develop into the adult worm in the intestine. The cæcum, colon and rectum are their favourite haunts, but they wander a great deal, and may be found at the anus, in the vagina or urethra, small intestine and vermiform appendix. Their number and frequency in this last situation has given rise to the suggestion that it may be their breeding ground.

Symptoms.—They closely resemble those of *Ascaris lumbricoides*, being often more severe and sometimes simulating appendicitis.

Treatment.—The standard treatment consists in enemata of either salt and water or infusion of quassia. Santonin is useful for destroying the worms in the cæcum and which therefore might escape the action of the enemata. Remember the risk of auto-infection and prevent the patient from scratching the anus. A weak mercurial ointment kills the worms which pass out and allays the irritation, and this should be regularly applied to the anus during treatment.

TRICOCEPHALUS DISPAR—THE WHIP-WORM.

This is another worm which inhabits chiefly the cæcum and colon. The body (female two inches, male one-and-a-half inches long) has its anterior three-fifths or more of a thin hair-like form coiled in a spiral manner and much narrower than the posterior part, which in the male is also spirally coiled but in the female straight and blunt. The eggs escape with the fæces but develop slowly, apparently

either outside or inside the body, in water and moist earth. They are oval, with ends shaped like buttons, and are about the same size as those of *Oxyuris vermicularis*. The embryos or the eggs may enter another host by the drinking water or by fresh green vegetables, such as lettuce. The worms are usually present in great numbers, and while chiefly met with in Paris and Germany are widely spread.

Symptoms.—There are none in the great majority of cases, but diarrhœa and severe anæmia sometimes occur and have been ascribed to this cause.

Treatment.—This worm rarely calls for very active treatment. It is probably most amenable to large thymol enemata.

ANCHYLOSTOMA OR DOCHMIUS DUODENALE —ANCHYLOSTOMIASIS.

The body is about one-third inch long, the female being slightly the larger. The mouth is on the ventral surface of the pointed head, and is armed with four tooth-like hooks for attachment to the mucous membrane. The posterior end is blunt, and in the male has a prominent bilobed bursa copulatrix supported by eleven chitinous rays. The eggs are oval, about the same size as those of *oxyuris*, and are also discharged with the fæces. They have a thin membrane, allowing the embryo within to be seen in various stages of development. The worms themselves do not usually escape except after vigorous purgation. Development and infection are much the same as in *tricocephalus*. The worms inhabit the duodenum, jejunum (chiefly) and other parts of the small intestine of man in most warm climates, especially Egypt and other parts of Africa, India, Ceylon, and Italy. It is not uncommon throughout Europe, being

spread by labourers coming from infected regions. Epidemics are thus apt to break out, as happened among the workmen employed in the construction of the St Gothard tunnel, among the brickmakers in Austria and Germany, among the miners in Belgium and France, and recently among the miners in Cornwall.

Symptoms.—They are very variable, but when any are present, which is usual, a progressive and severe anæmia is the chief. It is attributable to the parasites sucking the blood from the intestinal wall. It may be so profound as to closely resemble pernicious anæmia, from which, however, it differs somewhat in the blood changes produced, particularly in the hæmoglobin index being low and in poikilocytosis being slight. Debility and wasting follow the anæmia; but irregular fever, shortness of breath and œdema are also frequent. Locally the parasites may cause diarrhœa or irregular action of the bowels and colicky pains. The course is usually chronic but sometimes it ends fatally in a few weeks. It is noticeable that in African natives, in whom the worm is very common, it is the exception, and not the rule, for it to cause the prominent symptoms it induces in white people. This parasite belongs to the same family as the *Sclerostomum equinum*, which causes worm aneurysm in the horse, and is the only member of the family hurtful to man.

Treatment.—Thymol is the most efficient remedy. Give the patient, after a preliminary purge, 20 to 40 grains in wafer paper, and follow with a dose of castor oil. During treatment the diet must be milk and soups, and no alcohol should be taken, as it is a ready solvent of thymol and toxic effects may result. Several doses of thymol are often necessary, and thereafter the patient must be treated for the bloodlessness and debility so commonly present. Prevention is important, and care should be taken to

destroy fæces containing the ova, and to keep the water supply absolutely free from sewage, and more especially where the parasite has appeared.

TRICHINA SPIRALIS—TRICHINIASIS.

This worm in its adult state also lives in the intestine of man but differs from all the foregoing in requiring to inhabit an intermediate host during its development. The *adult worm* is cylindrical and thread-like, the female being about one-eighth inch long, the male half as much, and further distinguished by possessing two little projections near the anus. The *larval form* is that which is commonly seen. It is about $\frac{1}{30}$ inch long, pointed at both ends, except the female, which is blunt posteriorly. They lie usually singly, but sometimes in pairs or more, spirally coiled up within oval cysts, which when old are visible to the naked eye as white specks in the muscles. They are about $\frac{1}{70}$ inch long and have a laminated wall which is transparent at first but later opaque and infiltrated with lime salts. The larvæ in this encysted state keep alive for an indefinite period, though they remain sexually immature until the flesh containing them is eaten by another animal, when they become free in the intestine and reach sexual maturity in about two days. Union of the sexes takes place, and in a few days more (five or six) embryos begin to leave the body of the mother by the genital pore. Each mother may give birth to as many as a thousand embryos (Leuckart). These attach themselves to the mucous membrane, and after penetrating it wander through the peritoneum, &c., or are carried by the blood-vessels and lymphatics to the muscles, which they reach in the course of about a week, and settle within the sarcolemma. They quickly reach full larval

size, and the irritation due to their presence causes an inflammatory reaction within the muscle-fibres, which results in the formation of the cyst, which is generally complete in about four to five weeks. The cysts may be present in every muscle of the body in great numbers but are usually most numerous in the muscles of the abdomen, diaphragm, chest, larynx and tongue towards their tendinous attachments. Man is usually infected by eating pork containing the larvæ, the pig by the rat, and the rat by man. Cases in man are rare except where ham, wurst, &c., are eaten in a raw or underdone state, as in North Germany, but even there meat inspection is now so thorough that cases are uncommon.

Symptoms.—The worms in the intestine may cause pain, loss of appetite, nausea, vomiting or diarrhœa, sometimes very severe. These symptoms are often absent or slight, and when present last a week to ten days, and are followed by others caused by the migration and encystment of the worms. There is pain and stiffness in the muscles, fever, and œdema, first of the eyelids and face and then of the extremities. Sweating, wasting and anæmia appear, particularly in protracted cases, but the symptoms usually abate after the fourth or fifth week. In cases of intense infection the fever, delirium, dry tongue, tremors and general condition present a strong resemblance to typhoid fever.

Diagnosis.—Typhoid fever, acute rheumatism, acute tuberculosis, or cholera may be simulated by individual cases. Doubtful cases may be settled by the presence of an eosinophilia in the blood (the eosinophiles may number thirty-five per cent. or more of the total leucocytes instead of the normal two to four per cent.) or by finding the worms in the stools (they look like small silvery-white threads) or in a small piece of a muscle removed for examination.

Treatment.—If diarrhoea has not followed the ingestion of infected and uncooked meat, at once purge, and should there be time to empty the stomach before the contents have passed into the intestine give an emetic. Thymol might also be administered with advantage when the case comes under observation at an early stage; once the embryos have reached the muscles treatment must be on general lines, and nothing can be done to eliminate the encysted parasites. Pork should always be inspected, and especially pork which is intended to be used for food without actually raising it to boiling point.

DRACUNCULUS MEDINENSIS—THE GUINEA- WORM—DRACONTIASIS.

It is found in certain parts of Africa, *e.g.*, Guinea, Abyssinia and Egypt, and in India, Persia, and other warm climates. The male is doubtfully known as a degenerate worm, smaller than the female, which is one to six feet long and $\frac{1}{16}$ inch in thickness. Its (the latter's) habitat is the subcutaneous tissues of man, particularly of the feet and legs, where it causes vesicles or abscesses. It produces a vast number of eggs which develop viviparously and leave the body in the pus. The worm itself may also escape in the same way. In any attempt at artificial removals care must be taken to avoid injuring the body of the worm and so allowing the embryos to escape into the tissues of the host. The embryos seek stagnant water, and penetrating the body wall reach the body cavity of a minute crustacean (Cyclops). They then grow in size but do not become sexually mature until they gain entrance to the alimentary canal of man, which they do while still within their crustacean host. Impregnation then occurs and the female makes her way to the

subcutaneous tissues, where it attains its full development. The legs and feet are chiefly affected on account of the facility with which the embryos can reach the water after the skin is broken.

Symptoms.—The worm may remain quiescent for a long time, when it feels like a bundle of string beneath the skin. Usually embryos escape into the surrounding tissues sooner or later, especially if the worm be injured and an abscess results, which points through the skin; but if no embryos escape the worm may die in situ and become calcified.

Treatment.—On no account should the guinea-worm be wound out until the uterus is emptied of embryos, unless it be first treated by injecting the body of the parasite with 1 in 1000 corrosive sublimate, when it may either be wound or cut out. As the embryo probably enters the human subject in drinking water, care of the supply, so as to avoid contamination, should be insisted on; and it is safest to use boiled water for drinking purposes in countries where this parasite is common.

FILARIASIS.

Several varieties of filariæ are found in the blood of man, and are hence called *Filaria sanguinis hominis*. The best known are—1. *Filaria nocturna*.—It appears within the peripheral blood only at night, retiring to the pulmonary vessels and large arteries during the day. They begin to appear in the peripheral blood about five or six o'clock in the evening, increasing in numbers till about midnight, when as many as 300 may be found in each drop of blood, after which they gradually diminish, until they disappear altogether about eight or nine o'clock in the morning. If the patient reverses his

habits—sleeping during the day and working at night—the worms follow suit and appear during the day and disappear at night. The worm is about $\frac{1}{80}$ to $\frac{1}{100}$ inch long, and as narrow as a red blood-corpuscle. It has a blunt anterior and a pointed posterior end, and is enveloped in a structureless sheath which is larger than the animal, and so projects beyond it at one or both ends. The body of the worm is granular but shows no formation of internal organs. It is a larval worm and can undergo no further development within the blood, but can do so if withdrawn into the stomach of a mosquito. It is the female mosquito which acts as the intermediate host. After developing within the body of the mosquito for about a week, they escape into the water on the death of the mosquito, which happens when she seeks the water to lay her eggs. In 1900 Dr Low discovered an embryo in the proboscis of a mosquito and suggested that the worm may enter man when bitten by the mosquito in the same way as the malarial parasite. The embryos are generally believed, however, to enter the alimentary canal of man in the water, and make their way to the lymph-vessels, where sexual maturity is reached. The adult worm is called *Filaria Bancrofti*. It is common in all warm climates. The female is three to four inches long, brownish, and very thin (hair-like). The male is fully one inch shorter, transparent, and has a curled up tail.

Symptoms.—The *Filaria nocturna*, *i.e.*, the embryo, may exist for a long time without causing any trouble. So far as is known it gives rise to none, but the adult worm may cause trouble itself or by producing its young abnormally. When it dies it may cause an abscess, and when it produces eggs instead of free embryos, these eggs, which are thicker than the embryos and immobile, may get caught in and block the lymphatics. When the obstruction is partial a lymph varix is the result, showing itself as varicose lymphatic glands, lymph scrotum, chyluria, chylous ascites

or chylous hydrocele. When it is complete, rupture of the lymphatics ensues, and the lymph escapes on the skin (lymphorrhagia) or into an internal cavity or accumulates in the tissues, when swelling and hypertrophy occur, causing the condition known as elephantiasis of the limbs (Arabum) or of the scrotum. In chyluria the urine appears milky when passed, and on standing soon separates into two layers—an upper creamy coloured fluid, and a lower whitish or pinkish solid. Both layers contain fat in minute division and living filariæ. The condition is usually intermittent, disappearing sometimes for weeks or months, and may reappear quite suddenly. Fever, termed by Fayrer "elephantoid fever," usually accompanies all manifestations of filarial disease.

2. *Filaria diurna* is a larval form, similar to the *nocturna* except that it appears in the blood only during the day, commencing about eight or nine in the morning, increasing up to twelve and disappearing about eight or nine in the evening. It is found in the West Coast of Africa, in the lower Niger region. *Filaria loa* is thought by some to be the adult form. The male is shorter than the female, which is one-half to one-and-a-half inches long. This parasite inhabits the subcutaneous tissues about the bridge of the nose and of the conjunctiva, where it may cause great pain. Its geographical distribution is the same as that of the *Filaria diurna*.

3. *Filaria perstans* is also a larval form, much smaller in every way than the other larval filariæ. It has no sheath. It is always present in the blood, both night and day, although not in great numbers (three or four to a microscopic slide). It may be present along with either the *Filaria nocturna*, the *Filaria diurna*, or both. Its geographical distribution is the Congo district and other parts of the West Coast of Africa. For a time it was thought to be the cause of sleeping sickness, as it was found to be present in the blood of practically every

case of this disease. It has lately been shown that this relationship is accidental. It is claimed that a blunt-tailed species found in British Guiana is the parent form.

4. Other filariæ—*Filaria demarquaii*, *Filaria ozzardi*, *Filaria magalhæsi*—do not require separate notice.

Treatment.—Prevention is better than cure and the active destruction of the mosquito would go far to exterminate this disease. Chyluria, when present, should be treated by keeping the patient in bed for a long time, raising the pelvis, giving low diet, with a minimum of fat, and purging with salines. Elephantiasis may call for surgical treatment, but bandaging limbs affected, massage, and diminishing lymph pressure by position are worth trying. Many cases are incurable.

III.—THE ARTHROPODA.

This group is a very large one. It includes animals with jointed limbs. Very few are parasitic on man. Some of these belong to the arachnida and some to the insecta. The itch parasite—the *acarus* or *sarcoptes scabiei*—is the most important of the former. It is a turtle-shaped animal with four pairs of legs, the female being about $\frac{1}{10}$ inch long and $\frac{1}{100}$ inch broad. It is twice the size of the male. The female lives in burrows in the skin, the male only on the surface and is rarely seen. Each burrow forms a small whitish or whitish-black line and contains one female, which lives for two or three months and lays one or two oval eggs each day. The young are hatched in less than a week. The burrowing causes intense itching, and the consequent scratching causes skin excitations and inflammations. The delicate skin of the flexures of the body is usually attacked first, such as the webs of the fingers, the flexures of the wrists, elbows, groin, &c., but extension

to other parts of the body soon follows. The disease is usually called scabies or itch. It is easily communicated by clothing or by personal contact. Among the insecta the pediculi or lice are the commonest parasites. The pediculus capitis is the head louse. It multiplies with great rapidity. An adult female is said to produce 5000 eggs in two months. These eggs, called nits, are fixed to the hairs by a sort of split ring which allows them to be moved up or down but not detached therefrom. They are hatched about the sixth or seventh day, and the young become mature in about three weeks' time. In addition to the irritation caused by their presence and by scratching they often cause enlarged cervical glands in children. The pediculus vestimentorum is more elongated than the pediculus capitis. It inhabits the underclothing, making frequent excursions upon the skin for food, causing irritation, which is increased by the excoriations produced by scratching. The name phtheiriasis is given to this condition.

Treatment.—Scabies is best treated by inunction with sulphur ointment, and a preliminary hot bath with plenty of soap will help matters greatly. The same clothes should be worn night and day (next the skin) for three or four days, the ointment being applied nightly, and then a hot bath and clean garments should follow. Many other ointments are equally efficacious if not so commonly used, such as styrax (2 drachms to the ounce) or β -naphthol (1 in 10) in lard. Pediculi are usually associated with extreme want of cleanliness of person and clothing. The head louse may be treated by thoroughly rubbing into the hair paraffin oil, and then scrubbing the head with soft soap and water. Pediculus pubis should be treated in the same way only styrax soap should be preferred to soft soap.

IV.—*THE INTOXICATIONS.*

This is a convenient term for a group of diseases caused by poisons introduced into the body, in most cases from without, either with the food or as drugs. Strictly speaking it also includes poisons of microbic origin formed within the tissues as the result of the entrance into them of germs, and also poisons formed within the intestines or tissues as the result of perverted digestive processes or perverted metabolism, often spoken of as autogenetic poisons, such as those producing the conditions known as uræmia and cholæmia, and others as yet little known to us. The microbic poisons have already been considered. The autogenetic poisons will be briefly considered under the systems to which they seem most closely allied, and there remains therefore only the extrinsic group of poisons to be considered here. The most important of all is alcohol.

ALCOHOLISM.

It is true of all poisons, and particularly of alcohol, that the effect produced depends not only upon the dose but upon the personal idiosyncrasies, inherited and acquired, of the patient. This partly at any rate accounts for the wide variety in the results of taking it to excess. Death may immediately follow the sudden taking of a very large quantity of strong alcohol, but this is rare. Usually the patient becomes collapsed and sinks into a state of coma, due to the narcotic action on the cerebral nerve-cells. The skin becomes cold and clammy and the pulse small. The coma may deepen, and syncope and death, with or without convulsions, may follow, but much more commonly recovery takes place after a heavy sleep. When the dose is not so

large a period of excitement precedes the narcosis. The eye is bright, speech fluent, and the intellectual powers quickened, though the critical faculty probably becomes weakened from the outset. At any rate the intellect soon becomes clouded, the speech thickened, and the individual becomes drowsy. The movements, especially of the legs, become incoordinated. Drowsiness deepens into narcosis and he falls into a heavy sleep, to awaken little or none the worse for his indulgence, except for a headache, due to some imperfection in the liquor, or to consequent indigestion results. This is merely a bird's-eye view of the effects of *Acute Alcoholism*. The evil effects of alcohol are much more frequently seen after indulgence to excess over a long protracted period, spoken of as *Chronic Alcoholism*.

The symptoms of chronic alcoholism are of an extremely varied and often insidious character. There is often a gradual physical and mental deterioration, a nervous instability leading to indecision, irritability and loss of sleep. A fine tremor appears in the lips, tongue and hands, particularly brought out by muscular effort or a sudden start. Want of appetite appears, and like all the other symptoms it is worst in the morning. The tongue is generally dry and foul, the pharynx chronically inflamed, causing hawking and coughing, and the stomach similarly affected, producing nausea and vomiting. The bowels are deranged, often loose (a suggestive symptom) or constipated, or alternately the one and the other. The face is pale or shows signs of chronic venous congestion, particularly the nose, becoming later puffy and bloated looking, and the muscles become flabby and wasted, especially in the legs. The heart becomes feebler and the abdominal organs tend to become enlarged from venous congestion. Thus the liver and spleen may become palpable and tender, and albumen may appear in the urine. Cirrhosis of the liver, with its attendant symptoms of ascites, gastric congestion, &c., or the signs of multiple peripheral neuritis,

especially in women, may appear. The mental powers, sleep, and general nutrition become still more disturbed, until death, sometimes, however, not for many years, results through weakness or some lung, liver or kidney complication. *Delirium tremens*, commonly called "The Horrors," is a condition occasionally resulting from alcoholic poisoning, sometimes in acute cases but much more frequently in chronic. It is mostly met with in constant and hard drinkers — "the soakers." The attack may be brought on by an injury or an acute illness, especially pneumonia, or a bout of harder drinking than usual. A distaste for alcohol and a cessation therefrom is often one of the earliest symptoms. The victim becomes more restless and nervous and cannot eat. Sleep is fitful and disturbed by fearful dreams. Hallucinations and illusions occur, particularly at the time of waking from sleep. He talks a great deal. The stage of actual delirium now comes on. He may chatter incessantly, but generally answers questions intelligently. There is marked and constant tremor, affecting not only his hands, tongue and lips, but also his whole body. His face is pale and his eyes roam fearfully about. His mind busies itself with a whirl of ever-changing ideas, and he imagines that he sees and hears around him all sorts of fearsome things—swarms of rats, beetles, and terrifying crawling things. Later he ceases to recognise his friends and takes them to be enemies or fiends deriding him or seeking to poison or torture him. He is in a constant state of terror, will try to escape, and may attack his attendants, but can generally be controlled by firmness and tact. The temperature in mild cases usually rises to 100° or 101°F., and in severe cases to 103°F. After three or four days the attack wears itself out and the patient sinks into a quiet heavy sleep, from which he awakes free from delirium, or if not entirely so much improved, and a second sleep completes his restoration to sanity. He is very weak, but in favourable cases convalescence is rapid.

In severe cases death may ensue from syncope, from delirium, from exhaustion, or from convulsions.

Morbid Anatomy.—In acute alcoholism no typical lesion is to be found, but in chronic alcoholism there are many and important ones which may be grouped into—(1) Degenerations affecting (*a*) the secreting cells of the stomach, liver, kidney, &c., (*b*) the peripheral nerve-fibres, (*c*) the muscular fibres of the heart and systemic muscles generally; (2) fibrous tissue changes leading to cirrhotic changes in the liver, kidneys, stomach and nerve-fibres; (3) formation of fatty tissue, particularly subcutaneously, leading to obesity.

Treatment.—When an individual has taken an enormous quantity of spirit, thereby endangering his life, the stomach pump or a hypodermic of apomorphin ($\frac{1}{8}$ – $\frac{1}{6}$ grain) should be promptly resorted to. In most cases of occasional alcoholic excess the individual sleeps off the effects and needs no special treatment at all.

Delirium Tremens requires very careful management, although it is true that the condition runs a pretty definite course notwithstanding remedies employed. Sedatives are always called for during the stage of excitement, and there is much difference of opinion as to which remedies are the best. Opium or morphia and even chloral possess the great objection that they may initiate a new craving, and therefore they should only be used after due consideration, and should never be given freely. Prior to the administration of opium it is well to investigate the adequacy of the kidneys. Hyoscin ($\frac{1}{150}$ to $\frac{1}{100}$ grains) hypodermically is a powerful sedative, while a mixture of potassium bromide (40 grains) and chloral (20 grains) acts admirably, and may be repeated in an hour if necessary. Sulphonal, trional and paraldehyde are also good sedative remedies. Cold applied to the head is useful if there is fever, and

apomorphin administered hypodermically is very soothing in maniacal cases. Bed must be strictly enjoined, and a strait-waistcoat is most useful should the patient be violent, unless pneumonia or definite evidence of cardiac failure is present. Where a padded room is available and the patient very violent it is often of great service, and when the delirium subsides the patient may be put back into bed.

To support the strength is certainly the chief aim of the physician, and strong soup, eggs and milk should be freely given.

When heart failure is threatening, give caffein, spartein, or digitalis, the latter with caution. Try to keep the kidneys and bowels acting freely, and administer strychnin whenever the stage of acute delirium is past.

Chronic Alcoholism demands a different line of treatment. Dyspepsia and vomiting are common features, and may require the administration of pepsin, effervescing mixtures, and careful dieting. Sleeplessness, which may be a troublesome feature in acute alcoholism, is often a condition requiring careful management in the more chronic cases. Resort to hypnotics only as a last resource, and try first what open air, exercise, and massage will accomplish, together with such simple aids to slumber as a cup of soup just before attempting to court Morpheus.

But treatment of chronic alcoholism includes an effort at conquering the morbid craving for alcohol. The gold cure (chloride of gold, $\frac{1}{10}$ grain in 10 minims of water) administered hypodermically is a much vaunted remedy in the hands of those who provide "secret" cures for alcoholics. Its use is generally associated with strychnin and other nerve tonics. An attempt should be made to restore will power, and this is best accomplished by a residence of at least six to twelve months in a retreat. Hypnotism has been of service in some cases, but should be practised with great caution and only under medical supervision.

FOOD POISONING—PTOMAINE POISONING.

Poison may enter the body with the food, either in the form of (1) putrefactive changes therein before it is eaten, or (2) pathogenic micro-organisms of a special kind, *e.g.*, the *Bacillus enteritidis* of Gärtner and the *Bacillus botulinus* of Van Ermengem, infecting it and causing poisonous changes therein after it is eaten. In the former case it is obviously tainted to both taste and smell, but in the latter case it seems to be quite sound. Tinned foods, meat pies, sausages, &c., especially when imperfectly cooked, have been to blame in certain cases. The putrefactive processes are imperfectly known, but certain gases, acids and compound ammonias, known as the amines, are produced. The ptomaines, or animal alkaloids, belong to this last group, hence the name ptomaine poisoning.

Symptoms.—The time of onset is very rapid in the case of the first group, where the poisons are ingested with the food, whereas they do not come on at once but after a certain interval in the second group, where they are not ingested with the food but are produced afterwards within the body by the bacteria with which the food was infected. The symptoms vary considerably but may be arranged in three groups, any of which may be predominant in special cases—(1) Gastro-intestinal symptoms, *e.g.*, vomiting and diarrhoea; (2) cardiac symptoms, *e.g.*, depressed action of the heart, even to collapse; (3) nervous symptoms, *e.g.*, great prostration and exhaustion, convulsions or coma, and sometimes fever. Generally speaking, they mostly resemble either cholera or typhoid fever. They often prove fatal.

Ergotism is a special form of food poisoning, due to rye bread made of rye contaminated by the parasite *Claviceps purpurea*. It causes extreme contraction of the arterioles,

and simulates Raynaud's disease, but is now very rarely met with.

Pellagra is caused by the consumption of maize which has become mouldy. It is, like ergotism, a disease of the very poor, and closely resembles it in many respects. It is met with in Northern Italy, Southern France and Egypt.

Lathyrism is a similar but less severe disease, now only met with in India, but formerly also in certain poverty stricken districts of Southern Europe, and also due to diseased grain, probably certain species of vetch (*Lathyrus sativus* or *Lathyrus cicera*) mixed with the flour.

Mushroom poisoning.—Cases in which certain fungi are eaten in mistake for mushrooms are not uncommon. Poisonous symptoms may quickly set in, chiefly vomiting, diarrhoea and prostration from its great depressing action on the heart. In most cases the poison is muscarin.

Treatment.—The greatest care must be taken to inspect the carcasses of all animals to be used as food. No flesh should be sold for consumption from any animal which has died of any septicæmic disease. The chief risk is undoubtedly that unscrupulous butchers may sell such meat, either in the form of sausages or as smoked hams. Tinned meat should never be used if the contents of the tin smell at all putrid, or even if the jelly round the meat is liquefied.

When bad meat has been eaten the first thing to do is to get rid of the contents of the stomach and then to administer a dose of castor oil if the patient is able to stand it. Next try intestinal antiseptics, such as salol, β -naphthol, or sulpho-carbolate of soda.

If symptoms resembling muscarin poisoning have appeared try the effect of hypodermics of atropin ($\frac{1}{100}$ grain); if the symptoms rather resemble atropin poisoning then give morphia. Treat convulsions by bromides; faintness by stimulants—ammonia, strophanthus, &c.; and other symptoms as they appear.

Ergotism should be treated by feeding those who are affected with grain absolutely free from the fungus. Try the effects of warmth, stimulation, and gastro-intestinal sedatives, and amputate gangrenous extremities when necessary.

Pellagra and *Lathyrism* should be treated by eliminating the poison from the dietary, and treating the various symptoms as they present themselves.

Mushroom poisoning should be treated by removing from the stomach or intestine as much of the poisonous fungi as may have escaped digestion or absorption, and then endeavouring to antagonise the muscarin by giving atropin hypodermically and freely stimulating the patient.

SNAKE POISON.

The two classes of poisonous snakes are—(1) The vipers (adders, rattlesnakes, chain vipers) and (2) the colubers (cobras, kraits, coral snakes, moccasins, tiger snake and black snake). The poison is secreted by the parotid glands and belongs to the group of proteoses, similar to the proteoses formed by the *Bacillus diphtheriæ*. Much experimental work of a high and valuable character has been done by different workers in regard to the action of reagents upon venoms, to the effect of venoms when introduced into the living body, and especially by what means these effects can be counteracted after the introduction of the poisons.

Symptoms.—They vary much, according to the snake and the individual, the method of introduction, and the amount of the poison. Roughly speaking, they may be grouped in two divisions, according to the class of snake—viper or coluber—which is the most important of these

variable factors. In both cases the wound quickly swells and becomes painful, particularly after a rattlesnake bite. Then marked prostration follows. In the viperine group the affected part of the body becomes very oedematous and discoloured, the blood deteriorated from rapid destruction of the red cells, the urine albuminous, and the heart greatly depressed; whereas in the colubers there is little action on the blood, heart or kidneys, but great action on the nerve centres, particularly on the tongue, larynx and limbs, and later the respiratory centre, which become greatly weakened and even paralysed. In fatal cases death usually takes place in from six to twelve hours, though it may be much more rapid, or delayed for two or three days. In favourable cases recovery is rapid, the patient being all right again in a few hours.

Treatment.—Our first aim is to prevent absorption, and this is best attained by tying a ligature tightly round the limb above the part bitten. An attempt should next be made to suck out as much of the poison as possible, or by incisions at the site of the bite and then by forcible massage to squeeze out the venom. Caustics and antiseptics may be applied (nitric acid, 1% permanganate of potash, hypochlorite of lime), or the part cut out, or, lastly, in the case of the more poisonous snakes, amputation of a finger or toe is better than running a considerable risk of death.

If the poison has been absorbed we must try to antagonise its action in the body. Ammonia injected into the veins is one of the oldest remedies, but is probably much over-rated. Strychnin is often of very great benefit, and certainly alcohol should invariably be promptly administered. The researches of Calmette, Fraser and others have yielded an antidote of the greatest value, and antivenom, prepared in the Pasteur Institute and elsewhere, is capable of preventing death in some cases.

OPIUM POISONING—MORPHIA—MORPHINISM.

Acute Opium Poisoning.—This may arise accidentally, whereas chronic opium poisoning is always wilful. The symptoms of the acute form are due to the morphin present in opium. If a lethal dose be taken there may at first be some passing excitement, but coma quickly supervenes and death may, although not usually, result within an hour, but generally after some hours; the breathing, which is stertorous and slow, becoming more and more laboured, with signs of cyanosis about the lips. The pupils are contracted and the face of an ashen-like hue. Dilatation of the pupils may precede death, which takes place from paralysis of the respiratory centre. After smaller doses the symptoms are similar but less severe, according to the amount—if small, giving mere sleepiness; if large, coma of a more or less profound character.

Chronic Opium Poisoning (Morphinism).—The drug may be taken in either of three main ways, viz.—(1) Opium eating, in which the drug is taken in the crude state or in one of its crude preparations, such as laudanum; (2) opium smoking, in which sublimated products of the drug are inhaled; and (3) morphin injection, in which a solution of morphin is injected under the skin. The second is the least hurtful of the practices. The first is more prevalent among Eastern peoples. It is particularly common in India, and a remarkable tolerance for the drug is often established, so that very few of its injurious effects are noticed so long as steadily increasing doses are not taken. The third—morphin injection—is by far the most important of the practices. Within comparatively recent times it spread widely and became increasingly frequent, but now it fortunately seems to be less so.

Symptoms.—They are very variable, and in the absence of a confession it is very difficult to detect the early morphinist, but the discovery of the needle pricks on the limbs or body will often give the clue. Unpunctuality and general unreliability or disorderliness with plausibility appear to be among the earliest signs. There is often irritability of temper, an absence of moral rectitude, and later often a general failure in nutrition, leading to emaciation, a sallow, leaden or muddy complexion and contracted pupils. The bowels are generally confined, and all the secretions, except the sweat, diminished. Neglect of the duties of life increases, and he is only able to work when under the stimulating influence of a dose of the drug. Slovenliness is often marked, and later there may be great neglect of person. Intercurrent disease, particularly of the lungs, is very common, and sooner or later generally carries off the morphinist. If the drug be suddenly withdrawn from a morphinist he becomes restless and excited, is unable to sleep, his skin becomes clammy, he is often feverish, often deathly pale, suffers from diarrhoea, and may become delirious. Collapse sets in sooner or later and may become rapidly dangerous. These symptoms become relieved at once on the administration of a small dose, say one-tenth of the ordinary dose, of morphin hypodermically, and physicians are now generally agreed that gradual and not sudden withdrawal of the drug is the proper course to pursue.

Treatment.—*Acute Poisoning.*—Wash out the stomach at once, and failing a stomach-pump being procurable give an emetic. Then administer strong coffee, if necessary through the stomach tube. The patient must not be allowed to sleep, and should be walked about short of producing fatigue. Stimulants may be required, both alcohol and strychnin, and atropin is often a valuable agent. When necessary faradism should be applied to the

region of the heart, and artificial respiration may be called for, even for a period of two or three hours, aided by oxygen inhalations. The urine should be drawn off with a catheter because it certainly, if secreted fairly freely, contains a considerable amount of the poison, which would otherwise be re-absorbed.

Chronic Poisoning.—This is best treated by seclusion under conditions in which the patient cannot get the drug. Where an individual has been in the habit of taking huge quantities of morphia or other alkaloid of opium it is often better to diminish the dose gradually rather than to stop it too suddenly. Dyspepsia of a specially intractable description requires much painstaking treatment, while severe constipation necessitates the use of purgatives. Plenty of active exercise and healthy moral and intellectual influences are all of great value in expediting the process of cure.

COCAINE POISONING—COCAINISM.

Following the great use of cocaine by the surgeon and occasionally by the physician, especially in weaning a morphinist from his vice, a new habit as dangerous as morphinism, and as quickly acquired, known as cocaineism, has appeared, and is becoming increasingly prevalent. Cocaine is derived from the *Erythroxylum coca*, and it is true that the leaves have been chewed by the natives of Peru and other South American States from very ancient times, but the taking of the drug in the form of the alkaloid is a recent practice among civilised peoples, which has followed, as above mentioned, its use by medical practitioners. It is usually taken by the mouth, but sometimes by rubbing a solution upon the inside of the nostrils, and not infrequently by subcutaneous injection.

Symptoms.—They are much the same as those from morphin, a gradual ethical and moral, and later a physical, deterioration.

Treatment.—The depression which succeeds the temporary excitement requires careful study. Stimulants may be called for, sometimes alcohol but better ammonia. Artificial respiration is often necessary, with oxygen inhalations where the drug has been taken in great excess.

TOBACCO POISONING.

Only a few words need be said here about the over-use of tobacco. Ill effects from this cause are perhaps occasionally seen in even the most hardened smokers, who in consequence require to knock off or diminish their smoking for a time. They are by no means infrequent in a body of smokers physically or individually unable to become hard smokers when they indulge in what is an over-dose for them, although it might be trifling to others. Lastly, they are appearing more frequently in the present day among cigarette smokers, owing partly to the greatly increased prevalence of this habit, especially among immature youths, and to the very prevalent habit of inhaling the cigarette smoke into the lower respiratory passages.

Symptoms.—They are very variable, but they may be summed up in irritating or chronic inflammatory effects upon pharynx and stomach, cardiac flutterings and weakness, tendency to giddiness, and sleeplessness.

Treatment.—Great depression of heart demands prompt treatment with stimulants—alcoholic and other. In severe cases artificial respiration may have to be resorted to for a short time.

METALLIC POISONING.

Phosphorus, arsenic, lead and mercury are the most frequent examples of this group. Certain trades expose the workers to the absorption of small doses of these poisons, and, if this goes on for a sufficiently long period, injurious results ensue.

PHOSPHORUS—PHOSPHORISM.

It is the ordinary yellow form which is poisonous. The red variety is harmless.

Acute Phosphorus Poisoning.—Match heads, or phosphorus paste, or lumps of phosphorus may be swallowed. They give rise to symptoms within half-an-hour or more, according to the state of division of the phosphorus—the finer the division the more rapidly will the symptoms appear, and large pieces may pass through the intestine without doing much harm. The earlier symptoms are those of gastric irritation and are much less dangerous than the later constitutional ones, which appear three or four days after. These consist mainly in pain over the liver, which is found to be large and tender, though it may subsequently atrophy. Jaundice appears on the third to the fifth day. Vomiting is frequent. The pulse becomes feeble and the urine scanty. There is headache, insomnia and increasing weakness, and sometimes fever, particularly in the gravest cases. Drowsiness replaces the sleeplessness, coma generally supervenes, and death from cardiac failure after a course of generally under ten days.

Chronic Phosphorus Poisoning (Phosphorism).—This is principally met with in match makers, who are constantly

exposed to the phosphorus fumes. Anæmia, a yellow tint of the skin, and general ill-health come on. Phosphorus is detected in the urine and saliva. A still more serious condition is a necrosis of the jaw, almost always the lower jaw, known as "phossy jaw." It occurs chiefly in the "mixers" and "dippers."

Treatment.—For an acute case of poisoning, prompt administration of an emetic is necessary, and the emetic to give is copper sulphate (3 or 4 grain doses in a large quantity of water), or the stomach may be washed out with weak copper sulphate solution. In this way inert black phosphide of copper is formed in the stomach. Where a case of phosphorus poisoning, such as occurs in match factories, requires treatment, the following plan may be tried, namely—rigid milk diet, oxygen inhalations, and repeated small doses of turpentine. Try also to arrest phosphorous periostitis upon its first appearance in the jaws, using an antiseptic mouth wash; but actual necrosis is apt to follow, in which case nothing short of surgical treatment is of much avail.

ARSENIC POISONING.

Symptoms of chronic arsenical poisoning are apt to appear in those who take arsenic medicinally for long periods, or in those who are exposed to it in their trade, or clothes, or wall-paper, or beer, as was lately the case. They are gastro-intestinal irritation, anæmia and wasting, frontal headache, smarting of the eyes and eczematous eruptions in the skin, particularly where warm and moist, as in the axilla, upper part of thighs, and around the margins of the nostrils and eyes.

Treatment.—The administration of the drug should be stopped at once, or the source of the poisoning investigated and the patient removed from its influence. Potassium iodide helps elimination. Arsenical paralysis, when it develops, should be treated with massage and electricity, and also by strychnin administered by the mouth or hypodermically.

LEAD POISONING—PLUMBISM.

Chronic lead poisoning is the most important, frequent and subtle of the various forms of metallic poisoning. It occurs still with considerable frequency, though much less so than formerly, among workers whose trade involves the use of lead, *e.g.*, plumbers, painters, potters, enamellers, white lead makers, lead miners, smelters and melters, and among those who drink water contaminated by lead from pipes, cisterns, &c. The lead is either ingested with the food, often contaminated from the hands not being properly washed, or inhaled in the form of fumes by smelters and white lead makers, or in the form of dust by painters when rubbing down sized or painted walls, or, occasionally, by absorption through the skin or mucous membranes, as in the prolonged use of lead inunctions or plasters or certain hair dyes.

Symptoms.—Chronic lead poisoning occurs in four forms—

1. *The colic form.*—Colic is the most frequent complaint, no matter in what way the lead enters the body. It is spasmodic or continuous and usually very painful. It is usually preceded by anæmia and loss of appetite, and accompanied by obstinate constipation and pains in the

muscles. The colic may come and go. It is also accompanied by an extremely small radial pulse and oliguria, the secretion of urine being sometimes as low as eight to twelve oz. per diem. It contains too little urea. Other symptoms, seen not only in this form but in plumbism generally, are the pallor of the face, due in part to the general anæmia, and the appearance of a bluish-black line close to the margins of the gums. This latter is a sign of great value, and is best marked in those with bad and ill-cared-for teeth and gums. It is absent in those who regularly use the tooth-brush, though if once it does appear it cannot be made to disappear by this means. Its persistence is one of its most striking features, and it may persist for months after treatment has begun. It is due to the deposition of particles of lead sulphide within the tissues of the gum, probably from the sulphuretted hydrogen formed by the decomposing food, &c., around the bases of the teeth in dirty mouths acting upon the lead in the blood-vessels or tissues of the gums. Gout is another common ailment in plumbism.

2. *The nervous form.*—The central nervous system is greatly affected. There is headache or epileptiform seizures, called "lead encephalopathy." It is very dangerous and sometimes comes on without any warning. At other times, especially in young women, it is preceded by hysterical phenomena. The fit may last only a few minutes, but recurrences are frequent. If recovery occurs, the patient may be found to be partially or completely blind or aphasic. Colic or wrist-drop usually precedes the cerebral symptoms.

3. *The neuro-muscular form.*—Wrist-drop is the most characteristic feature in this form. It is due to paralysis of the extensors of the wrist and thumb, and is generally symmetrical, though one side is generally worse than the other—the right in right-handed persons. It is quickly followed by atrophy of the muscles which give the reactions

of degeneration. The supinator longus escapes, and this is an important guide in diagnosis. There are often other forms of peripheral paralysis, and it may be widely distributed. It may gradually disappear or persist. Colic occasionally precedes the wrist-drop.

4. *The cachectic form.*—The patient is extremely cachectic. The malnutrition is great. There may have been attacks of colic, wrist-drop and other paralysis. The urine contains albumin, and weakness is great. The individual ages quickly and prematurely, and dies from some intercurrent disease.

Treatment.—The preventive measures for lead poisoning include the disuse of lead piping for the water-supply in connection with houses, and especially where the water is soft, or from even slight contamination a good solvent of lead. Lead may be protected by the addition of carbonate of lime to the water, but under no circumstances should water which has been “standing” in the pipes be used, it should invariably be run off before a supply for drinking or cooking purposes is taken.

In factories where lead is much used, attention to cleanliness, especially before meals, and in certain industries the use of respirators, have done much to reduce lead poisoning to a minimum.

In the treatment of lead poisoning, potassium iodide in five to ten grain doses along with the free use of magnesium sulphate as a morning purge are much vaunted remedies. Lead colic calls for hot fomentations, and, if severe, the careful administration of opium. Lumbar puncture, with the removal of one to two ounces of cerebro-spinal fluid, has been used with success in treating severe encephalopathy. Lead palsy should be treated with strychnin internally, or better by hypodermic injection, together with massage and appropriate electrical applications, and the anæmia combated with iron and possibly bone-marrow.

SUNSTROKE.

Syn. Insolation, Heat Exhaustion, Thermic Fever.

Etiology.—These names are not quite synonymous, for while Sunstroke refers to exposure to the direct rays of the sun, Heat Exhaustion refers to prolonged exposure to high temperatures, whether in the sun or in the shade, *e.g.*, stokers in the stoke-holes of steamers. Troops who have to do long marches or manœuvres in the sun are liable to sunstroke, as was illustrated at Aldershot in 1901, when several soldiers died from it. The normal mechanism for regulating the temperature of the body (see article on "Fever") is upset, and the vaso-motor centre in the medulla becomes paralysed. It is not so obviously an "Intoxication" as the preceding conditions, but the analogy is sufficiently close to justify its position in this group.

Symptoms.—In either of the forms the patient may be struck down suddenly and die within an hour from heart failure. In heat exhaustion he generally becomes faint and pale, his skin moist, his pulse feeble, and he staggers and faints. In insolation proper there is giddiness, nausea, a high temperature (104° , 106° , or even 110°F.), restlessness, breathlessness, the pulse rapid and full, the pupils at first dilated then contracted, then unconsciousness and coma. Death may follow within $\frac{1}{4}$ hours, from paralysis of the respiratory centre. In other cases (the majority) the fever falls, consciousness returns, and recovery is rapid and complete. Sometimes, however, curious after-effects remain, the most striking and constant being an intolerance of the sun's rays if at all hot, or even of any temperature above 80°F. or thereabouts.

Treatment.—In cases of heat exhaustion, free stimulation must be promptly resorted to (alcohol, ammonia, and even the hot bath if temperature be subnormal).

In sunstroke proper there is generally a very high temperature, and this should be reduced by douching with cold water, ice pack, or ice-water enemata. Occasionally, when cyanosis is a special feature, venesection is called for.

In all cases of heat exhaustion or sunstroke remember the great necessity for promptness in treatment.

V.—GENERAL DISEASES OF OBSCURE ORIGIN.

RHEUMATIC FEVER—ACUTE RHEUMATISM.

Lat. *Rheumatismus acutus*. Fr. *Rheumatisme articulaire aigu*.

Ger *Acuter Gelenkrheumatismus*.

DEFINITION—A specific febrile disorder, characterised by non-suppurative inflammation of the joints and of the fibrous tissues surrounding the joints, of which many are affected at the same time, or in succession.

Etiology.—Rheumatic fever, though universal in its distribution, is most frequent in moist temperature climates, such as England, in which it is particularly prevalent. The greatest number of cases occur in October, November, and December. Young adults are most frequently attacked, but it is not uncommon in children. Males are more frequently affected than females, except before the age of 15, when the reverse holds good. Heredity is held by many to be an important predisposing cause, and the evidence in its support is considerable. A chill or exposure to inclement weather conditions was credited of old as being sufficient in itself to produce the disease, and it is remarkable how

frequently they are associated. The same is true of lobar pneumonia, but we no longer give all the credit to the chill. This is now known to act as a predisposing cause only, while the true exciting cause is the pneumococcus. Without the chill, the organism would no doubt in many cases be powerless to set up the disease. So with rheumatic fever the influence of the chill may be great, but it can act only by reducing the resisting power of the tissues, and thus enable the true exciting cause to do its work. We are still ignorant of the true nature of this exciting cause. From the close analogies presented by the disease to the acute infectious diseases of known bacterial causation, the microbic theory of its origin is a natural one, and it has received many supporters. Much work has already been done, and several bacteria have been described in association with the disease. The results of this work have so far been inconclusive, and may be summarised as follows:—(1) The disease is due to an organism still unknown; (2) it is caused by any or all of the bacteria of ordinary inflammations—*e.g.*, streptococci and staphylococci—and not by a specific organism; (3) it is caused by a specific bacterium—a bacillus according to some, a diplococcus according to others. There is considerable evidence in favour of the diplococcus of Poynton and A. Paine.

Attention has for a considerable time been directed to the tonsils as a point of entrance of the germ, on account of the frequency of tonsillitis as an initial symptom in the disease. There are two other theories which have been advanced to explain the origin of the disease which must be mentioned, *viz.*, the chemical, or metabolic, and the nervous. According to the former an aberrant chemical substance produced by some fault of metabolism gains entrance into the blood and sets up the disease. Lactic acid or certain of its combinations have been blamed, but the evidence is incomplete. According to the nervous

theory the disease is due to trophic mischief, traceable to the changes produced by the chill in the nerve centres, or to abnormal metabolism caused by the nervous disturbance.

Morbid Anatomy.—There are no special changes. The affected joints or serous membranes show ordinary inflammatory changes. The fluid from the joints is clear or turbid, and contains leucocytes and fibrin, often in considerable quantity, but it is not purulent. The blood shows marked diminution of the red cells, leucocytosis, and contains an excess of fibrin-forming constituents, like that of pneumonia.

Symptoms.—These vary considerably in the child and in the adult. In the latter the disease may set in gradually or abruptly with a feeling of chilliness, occasionally with a distinct rigor, or there is malaise and general weakness and pains in different parts of the body, especially the limbs. Fever quickly appears and the temperature rises rapidly to almost 102 or 104°F. The pains at the same time become prominent in one or more of the joints, which soon become swollen and very tender. It is usually the larger joints—the wrist, elbow, knee, ankle, &c.—which are primarily affected, but there is no rule or symmetry about their invasion. The swelling is considerable, but the skin over the joint is rarely red. The effusion is mainly within the cavity of the joint, but the periarticular tissues, and sometimes also the neighbouring tendinous sheaths, are implicated. The pain is frequently excruciating, and is increased by the slightest movement. Another striking feature of the joint affection, which, although liable to great individual variations, is always a prominent feature of the attack in adults, is its tendency to flit from joint to joint, the inflammation subsiding in one joint as it invades and increases in another. Thus within a few hours both the swelling and the pain may have practically

left the elbow and broken out in the knee. This, however, does not always happen, and several joints may be severely implicated at the same time. The skin is usually bathed in a copious sour-smelling acid perspiration. The temperature has no distinctive range but has many ups and downs (resembling a septic curve), generally between 101 and 105. Occasionally there is a hyperpyrexia (a very grave symptom), the temperature rising to 106°, 107°, 110°F., or even still higher. The ordinary rises of the temperature are usually coincident with the outbreaks of the joint inflammations, and falls thereof with their subsidence and the onset of profuse perspiration. The pulse is rapid and full but regular. The alimentary (apart from the tonsillitis) and urinary systems show the usual febrile phenomena. The circulatory system is implicated in a very large proportion of cases, probably about 50 per cent. of adults and far more in children. The commonest lesion is endocarditis, the mitral valve being most frequently attacked. The endocarditis may be simple or ulcerative and may supervene suddenly or gradually. The heart should be examined daily throughout the course of the disease, and frequently during and after convalescence. Pericarditis is the next commonest lesion, but myocarditis is not infrequent. The respiratory system shows pleurisy, especially left-sided, not uncommonly, and pneumonia occasionally. Iritis and certain skin affections, such as sudamina, erythema nodosum, and urticaria, occur. The disease has no regular course and gradually subsides in from eight to twenty-one days, or even longer, though the average duration under appropriate treatment is generally under three weeks. It must be regarded as a general disorder due to a toxæmia of the whole system, of which the joint, cardiac, and other lesions are parts of the process, being local manifestations, and not true complications, and naturally subject to considerable variations in individual cases. In children, however, the variations are so great and so uniform that they merit special notice.

It is held by some authorities that the disease as exhibited in children is more typical of its nature and course than as seen in adults. The joint affections are often very slight and may easily escape notice, although they may be marked in severe cases. The fever is generally slighter. The circulatory lesions are generally much more frequent and grave. The heart may be affected in the slightest cases. An acute dilatation is very commonly, if not always, present, and endocarditis, pericarditis and myocarditis are all more frequently seen than in adults. The main incidence of the disease appears to fall upon the heart, whereas in adults it is upon the joints. Two other distinguishing features require special mention, viz., subcutaneous nodules and chorea. The nodules are firm and fibrous, painless, though sometimes tender, and slightly movable, and are found over the ends of the long bones—*e.g.*, elbows, knees, &c.—the spinous processes of the vertebræ, or any prominent bony points. They are present in about a quarter of all the cases, particularly in the offspring of rheumatic parents. They vary from the size of a pin's head to that of a pea, and are either many or few in number, appearing simultaneously or in crops. They are often large or numerous in severe cases where the heart is seriously implicated. Chorea is another frequent manifestation of rheumatism in childhood, though the exact relationship between the two diseases has not yet been cleared up. It may precede or be coincident with, or follow the rheumatic attack. This varying sequence and combination is not unusual, being met with in the various manifestations of rheumatism in childhood.

Relapse.—If any immunity is established it must be of a very transient kind, as an attack seems to predispose the patient to a subsequent one; three or four or more recurrences in the same patient being not uncommon. These recurrent attacks may be due to fresh infections, or to a recrudescence of the first infection after a period of quiescence,

and with each recurrence the tendency of the poison to attack the heart would seem to be increased.

Diagnosis.—In typical cases the diagnosis is easy, more so in adults than in children. There may be difficulty at times in distinguishing it from certain other diseases, such as gout, gonorrhœal rheumatism, rheumatic arthritis, and arthritis caused by pyæmia or nervous lesions. A careful examination into the history and all the features of the case will generally enable this to be done. The tendency to metastases if present often suffices, as none of the other diseases mentioned exhibit it to any degree. The difficulty will most frequently arise between the monoarticular forms of acute rheumatism and gout, and then attention should be paid to history, age, size of joint, remissions, condition of nutrition of patient, and presence or absence of tophi and uric acid in the urine. Gonorrhœal rheumatism is due to the gonococcus, is very obstinate, and does not yield to salicylates so long as any urethral or vaginal discharge is present.

Prognosis.—Graver in children than in adults on account of the greater frequency of cardiac affections. In the former the mortality is greater than in the latter, in which it is very slight—less than three per cent. This applies to the rheumatic attacks apart from the cardiac affections, which may not manifest themselves till long after the direct attack of the fever has disappeared.

Treatment.—The first duty of the medical adviser is to have the sickroom suitably arranged. The bed should be prepared with a fairly soft, but not a spring mattress, and the patient must lie between blankets to prevent the sweat from chilling down the sufferer, which it would inevitably do were sheets used. The nightdress should be of soft flannel; slit down the front and sleeves so that it can be

changed, when damp, without moving the patient to any extent. Absolute rest is enjoined, but as the invalid is generally too pained to wish to move, this injunction is almost superfluous. The diet is confined to milk food, but if this cannot be taken then simple soups are substituted. Plenty of alkaline mineral waters can be given, and drinks such as lemonade, imperial, and barley water may be freely permitted.

The special medical treatment resolves itself into two parts—(1) Internal and (2) Local.

I. INTERNAL.—The old line of treatment, in which much faith is still reposed, is the alkaline method. It was supposed to counteract the excess of acid in the blood, upon which at one time the presence of the disease was thought to depend. It is still believed to diminish the coagulable tendencies of the fibrin of the blood, and so prevent the formation of depositions on the heart valves. Sodium or potassium salts are both used, perhaps the bicarbonate of the latter being most favoured in doses of about 10 to 30 grains thrice daily. The more modern method of treatment is to use the salicyl group. Sodium salicylate in 20 grain doses may be administered every four hours till the pain abates, and then less frequently; or else salol or salicin in similar doses may be substituted. It is of great advantage to combine with the member of the group used some bicarbonate of soda or potash. It has been urged that the sodium salicylate is dangerous, or at least depressing, while the toxic action of the drug may manifest itself most unpleasantly. It nearly always causes some cinchonism, some depression, and it may produce delirium in a very few cases. Lastly, a trifling erythematous rash may develop in some patients who have a peculiar idiosyncrasy to the drug, and this may alarm the patient or attendant. These evil effects can to a great extent be prevented or reduced to a minimum by the preliminary administration of a purgative, such as a dose of

calomel or a saline cathartic. Hydrobromic acid is also of use in counteracting the tendency to cinchonism. The synthetic salicylic acid should never be employed, as it is much more toxic than the acid prepared from vegetable sources (probably from the presence of paracresolic acid). It is worthy of remembrance that in cases where pericarditis ensues it is wise to stop the sodium salicylate, and in the event of heart failure threatening, such a depressing remedy is absolutely contra-indicated. In such cases it is best to substitute salicin, which is much less depressing, and in the treatment of children it should always be preferred to the soda salt. There seems no reasonable doubt as to the great value of this specific treatment of acute rheumatism, and that if commenced early and carried out conscientiously heart disease is often prevented or at least kept in check.

Pain may be relieved by the administration of an occasional dose of Dover's powder, and where absolutely necessary phenacetin or one of the other members of that group may be given. Cardiac tonics and diffusible stimulants are not, as a rule, desirable or necessary, but where the heart is severely affected, either by endocarditis or myocarditis, alcohol may be urgently called for, and must be administered.

Anæmia, which so often follows a severe case of rheumatic fever, should be treated in the usual way—with iron and other tonics.

Hyperpyrexia must be dealt with promptly by cold sponging, cold affusion in a tepid bath, or the application of iced cloths to the abdomen. These methods have been fully described under the treatment of the fever process generally.

2. LOCAL.—The affected and painful joints may be treated in various ways. They may be merely wrapped up in cotton wool, and they are sometimes placed in splints, so as to prevent movement during sleep (which may make the sufferer wake with a start), and ensuring in any case absolute rest. A better plan is to put on a preliminary

linseed meal poultice, a liniment such as Fuller's lotion (sodium carb. \mathfrak{zvi} , laudanum \mathfrak{zviii} , glycerine \mathfrak{zxvi} , water to 9 oz.), or to rub in a more vigorously counter-irritating liniment, such as linimentum terebinthinæ aceticum. One of the more recent local applications is salicylate of methyl applied on lint, and covered over with indiarubber tissue, which may be sealed down with chloroform at the edges, so as to keep in the smell, which is nauseous and penetrating. The salicylate of methyl is absorbed in a short time, and has proved successful in not a few cases. Blisters above and below the joint find favour with some authorities, or what is perhaps better still, light applications of Paquelin's thermo-cautery. Personally, we must plead for the frequent application of iodine (Edinburgh tincture alone, or the tincture and linimentum iodi in equal parts), especially after the very acute stages have passed off, as being one of the very best local applications which can be used in this disease. Lastly, we must refer to Caton's recommendation of small blisters applied repeatedly along the course of the second, third, and fourth dorsal nerves on the left side; this he considers prevents the development of cardiac disease, and he has obtained satisfactory results from carrying out this device. Dr Caton keeps the patient so treated in bed for six weeks.

CHRONIC ARTICULAR RHEUMATISM.

Etiology.—This disease may follow upon acute or sub-acute rheumatism or occur independently. While it follows upon, it is not a part of the acute or sub-acute disease but a sequela thereof. Acute inflammation of a joint is a common feature in acute rheumatism, this acute inflammation may be subsequently followed by chronic fibroid

changes in or around the joint, which, although they are the direct result of the acute inflammation, are not in any sense a part of the original acute rheumatism, which itself never becomes chronic. Chronic articular rheumatism is an entirely different disease from acute articular rheumatism. It is true, as already explained, that it may follow upon it; but it may also follow other diseases, such as influenza, or arise insidiously. It is most frequent in old people, a slight degree constituting the "stiffness of age," but it is also common in adolescents and in early middle life. Considerable destructive changes in the proper structures of the joint (cartilage, bone and synovial membrane) have been described by some authors, but most of them now agree that the proper joint structures usually if not always escape, and that the nodular and diffuse thickenings characteristic of the disease involve the structures around the joint, viz., the ligaments, tendinous and nerve sheaths, and fibrous tissues generally. Cardiac lesions are rare except in cases which follow upon acute articular rheumatism. They never follow upon chronic articular rheumatism itself and their co-existence therewith merely means that both lesions have a common origin.

Symptoms.—There is pain, stiffness, difficulty, and sometimes audible grating, in moving the joints. One or more joints, generally the larger ones, may be affected, and recurrent attacks, with intervals of comparative good health, are frequent; but the patients often complain of permanently feeble health.

Treatment.—Most patients require somewhat different treatment from that used in acute cases, depending much upon the severity and chronicity of the disease. Internally the salicyl group is generally only of benefit where a slight exacerbation has occurred, and it is better to give potassium iodide (5 to 15 grains thrice daily) along with guaiac resin (5

to 10 grains in capsules). Sometimes quinine (3 to 5 grains), which is recommended by Garrod, associated with an alkaline treatment, is very efficacious. Externally, counter-irritation is of great value; blisters, iodine (tincture and liniment in equal parts), the Paquelin thermo-cautery or the button cautery, and various liniments act in this way. Massage and passive movements, especially where muscular atrophy is present, are useful and prevent ankylosis. Baths of many kinds are beneficial, and especially radiant heat, electric, Turkish and hot alkaline baths, also peat baths and hot baths with the under-current douche. The wet pack applied to joints has met with success in some cases. Lastly, we should enjoin warm clothing during cold weather and if possible send our patients to a warm, dry climate during the damp and inclement seasons at home.

MUSCULAR RHEUMATISM.

Etiology.—The causation of muscular rheumatism is obscure; but there is strong evidence for looking upon it much in the same way as the last disease, muscular and articular rheumatism having much in common, both in causation and in morbid changes. In both, the essential change consists in thickenings of the white fibrous tissue; but their distribution is different. In the one case they are around the joints while in the other they are around the muscles. Moreover, in many instances they are combined, and there may be thickenings apart from either the joints or the muscles, as in the front of the sternum.

Muscular rheumatism may be acute or chronic. It is especially common in certain regions of the body, *e.g.*, back (lumbago), chest (pleurodynia), and neck (stiff neck); the muscles being swollen, stiff and painful, especially on

movement. There is generally no pain in the position of rest; but on any sudden movement it occurs, often so severely as to make the patient cry out.

Treatment.—The intense pain from which the patient may be suffering calls for very prompt treatment. Internally it is well to try one of the salicyl group along with an alkali, and if that fails, or the condition be more chronic, iodide of potash (in 10 to 20 grain doses thrice daily) and guaiac resin (10 grains in capsules) should be given.

Local treatment yields the quickest relief where the muscular rheumatism is very severe, whether it be pleurodynia, lumbago, or in some other region. Poulticing, applying dry cups over the painful muscle, the application of turpentine and other liniments, or even blisters, and the thermo-cautery are all efficacious. Ordinary hot baths, Turkish and steam baths, are most soothing, and many varieties of baths, such as electric, brine and mud, have all their supporters. Rest to the affected part, especially in cases of pleurodynia, is best obtained by strapping. In a few very obstinate cases the introduction of a few acupuncture needles into the affected muscles has proved in our hands of great benefit.

GONORRHŒAL RHEUMATISM.

DEFINITION.—The commonest form of gonorrhœal infection when it has become generalised; characterised by an effusion into a joint or tendon and the capsular structures around of a very obstinate character.

Etiology.—It comes on at any stage of a gonorrhœal infection, sometimes early (a few days after the first appearance of the urethral discharge), or more commonly later (some weeks after), either when a chronic urethral discharge

is still present or after it has disappeared. It is due to the invasion of the joint or tendon by the gonococcus, which reaches it by the blood-stream. The preliminary circulation of the gonotoxin may play an important part in preparing the ground for the germ. This absorption of gonotoxin to a greater or less extent must take place in all cases of gonorrhœa, but in the great majority the germ itself does not spread beyond the primary urethral site, the defensive powers of the blood and tissues preventing it from doing so. In other cases, however, it does spread, either by local extension—causing cystitis, endometritis, salpingitis and peritonitis—or by the lymph- or blood-stream. Lymphadenitis (bubo) is generally due to a mixed infection. By the blood-stream the germ may reach a joint (most common), a tendon sheath, a fascia, the eye, the heart, pleura or meninges, the muscles, nerves or arteries. In regard to the eye, when the conjunctiva first suffers, the infection is probably always direct, as in the case of infants at birth, whereas when the sclerotic or iris becomes affected during the course of an arthritis, the infection is probably by the blood-stream.

Pathology.—The serous tissues of the body are the special seats of selection, thus we get an arthritis, synovitis, conjunctivitis, scleritis, pleuritis, pericarditis, endocarditis, meningitis and peritonitis. Not only tendons but muscular fasciæ may become inflamed, particularly in the back, thighs and soles of the feet. Joints, as already said, are the commonest examples of general infection, and until comparatively recent times were practically the only known sites of the generalised disease, hence the long established use of the term gonorrhœal rheumatism, while gonorrhœal endocarditis, meningitis, &c., are modern terms. Any joint may be affected, but the knee-joint is that which is most commonly chosen, the ankle and wrist coming next, and then the elbow, the joints of the hands and feet, and the

sterno-clavicular, temporo-maxillary, and sacro-iliac, in the order named. These three last are hardly ever affected in other forms of joint mischief. The arthritis is most frequently single, but it may be multiple, and is often symmetrical. The fluid is serous; when purulent the infection is mixed, and not only fills the cavity of the joint, tendon, &c., but also infiltrates the capsule and fibrous tissues around. The inflammation is obstinate in character, but when once it has subsided it does not tend to recur.

Symptoms.—There is swelling and often redness of the affected joint or part. It generally comes on suddenly, and is painful and tender, particularly when it appears early in the disease, when there is also some general disturbance, such as fever. On the other hand, when it appears later there is usually no general disturbance, and the pain, if present, may be little more than dull aching. After it has subsided, which may not be for many weeks, it often leaves a good deal of stiffness, even amounting to ankylosis, due to adhesions and thickenings about the joint.

Diagnosis.—The history, the character and persistence of the joint trouble, and the presence of catarrhal ophthalmia are the most salient guides in making a diagnosis.

Prognosis.—It is generally very favourable, though the tendency to stiffness and to the implication of other serous tissues must be borne in mind.

Treatment.—Perhaps the only drug of special value is potassium iodide in 5 to 10 grain doses. Rarely do the salicylates afford much, if any, relief.

The chief attention should be paid to the joint affection and to the general nutrition of the patient. Hot air baths, blisters, iodine and other similar treatment are of great value; and some such remedy as iron, malt extract and

cod-liver oil should invariably be administered. Occasionally surgical interference is called for, but it is rare that more than moderately rapid improvement follows even the very best treatment in this disease.

ARTHRITIS DEFORMANS OR RHEUMATOID ARTHRITIS.

Lat. *Osteo arthritis*. Fr. *Arthrite sèche* Ger. *Deformirende Gelenkentzündung*.

DEFINITION.—An acute or chronic affection, characterised by pain, stiffness and deformity of one or more of the joints, associated with destruction of the cartilages, and a deposition of new bone around them.

This disease is known under a large number of synonyms, such as osteo-arthritis, arthritis nodosa, chronic rheumatic or rheumatoid arthritis, rheumatic gout, arthritis deformans, rheumatoid arthritis; though the last two are in most common use none of them are free from objections. The names suggest a close connection with rheumatism on the one hand and gout on the other, still believed by many to exist, though recent observations tend to disprove it.

Etiology.—1. AGE.—The chronic cases are found mainly in middle and advanced life, the acute cases mainly in the young.

2. SEX.—The chronic polyarticular form affects women much more than men, but the chronic monoarticular form men more than women. The acute form affects the sexes almost equally.

3. HEREDITY.—There is a considerable amount of evidence to show that a family history of joint trouble

predisposes to this disease, and phthisis is stated to act in the same way.

4. RHEUMATISM AND GOUT.—In Garrod's analysis of 500 cases there was a history of gout in the family in nearly one-third, and of rheumatism in 64. Cases following closely after both rheumatism and gout are not infrequently observed, but most authorities now believe that, whatever influence these diseases may have in favouring the onset of arthritis deformans, they are not in any sense identical, but essentially different, diseases. In the same way it may follow in joints that have been formerly affected with other mischiefs, such as gonorrhoeal rheumatism and traumatic arthritis.

5. Injury, cold, worry, insufficient food, and an enfeebled vitality may all act as more or less important factors in setting up the disease, whether as predisposing or exciting causes it is difficult to say in the imperfect state of our present knowledge of its causation. The two chief theories of its etiology are the neural and microbic.

6. THE NEURAL THEORY.—Joint lesions, both acute and chronic, are well known in lesions of the central nervous system, *e.g.*, infantile paralysis, locomotor ataxia, &c., and the suggestion that arthritis deformans arises from some disturbance of nutrition springing from some change in the nerve centres of the cord is a natural one. No change has as yet been demonstrated in these centres, but the analogies to other joint lesions of proved nervous origin, the frequently symmetrical onset and course of the disease, the dystrophies of skin and muscles, and also often of nail and bone, all suggest that these nerve centres are to blame.

7. THE MICROBIC THEORY.—Several observers (Schüller, Bannatyne, Wohlmann, Blaxall, &c.) have found a very small bacillus, showing bipolar staining, constantly present in the fluid and tissues of the joints in acute cases. Pure cultivations have been made, but experimental inoculation has not succeeded in reproducing the disease. The bacillus

has not been found in joints affected with other diseases, but the evidence in its favour has not yet been accepted as complete by bacteriologists. Whether it be this or some other bacillus, it does not exclude the nervous system from playing an important part, since the bacterial toxins may primarily affect the nerve centres through the circulation.

Morbid Anatomy.—The disease begins in the cartilage. Its cells proliferate and the matrix softens and becomes fibrillated, thus assuming a velvety appearance. About the same time the synovial membrane also becomes thickened. Its cells proliferate and its fringes enlarge. In acute cases, it becomes soft and vascular, but in chronic cases hard and pale. The synovial fluid is increased in all but the most chronic cases. Meanwhile, the softened cartilage at the centre of the articular surface gets gradually worn away until a varying part of the surface of the bone is laid bare. In acute cases this exposed bone is soft, red, and eroded, but in chronic cases hard and condensed, smooth and polished like ivory. Below the surface the bone is rarefied, absorbed, and atrophied, giving rise in time to shortening. At the periphery of the joint, where the pressure is less, there is overgrowth of cartilage, in which, as well as in irregular deposits of cartilage which may form within the thickened synovial fringes, ossification occurs, forming irregular bony masses and outgrowths (osteophytes) which enlarge the ends of the bones, producing the “lipping” felt during life. The periosteum may also form new bone, &c. In acute cases this new formation of bone is but slight, in chronic cases it varies greatly, being often very great. The ligaments in acute cases are swollen and inflamed and sometimes absorbed, causing dislocation and extensive disorganisation of the joints; in chronic cases, on the other hand, they are hard and thickened and fused with the other periarticular tissues; the joints are consequently so locked together as frequently

to be completely immovable, but true ankylosis does not occur except in the vertebræ. In the monoarticular variety—*e.g.*, the shoulder or hip in old people—the shortening from absorption of the cancellous structure of the head of the bone is often very great; but the locking is rarely so great as to entirely prevent useful movement. Though the disease in these cases is almost always limited to one joint, it may exceptionally spread to other joints. Another form of the disease causes small bony outgrowths (Heberden's nodes) from the third and second phalanges, close to the articular surfaces, and covered by an outgrowth of the synovial membrane. There is very little erosion of cartilage, and the process is very chronic, rarely extending to other joints, and becoming severe. In all forms but the last there is pronounced wasting of the muscles, and peripheral neuritis has been found in a few cases, probably accidental, in the nerves about the joints. In many of the acute cases in children the lymphatic glands of the extremities and the spleen were found to be enlarged.

It is possible that the disease, as above described, may include several separate diseases, thus the polyarticular may come to be shown to have a different causation from the monoarticular form, but at the present time no separation can be made.

Symptoms.—In the acute form some of the joints, usually the fingers, begin to swell. This may or may not be preceded by a sensation of tingling or pain in the joints or muscular eminences (thenar and hypothenar) of the hand, &c. The pain is generally pronounced after the joints become swollen, though redness is not frequent. Other joints become affected rapidly and with a marked tendency to symmetry in distribution. Destruction of the joints is rapid, and in a few weeks the patient may become a helpless cripple. There is some fever, much atrophy, and frequently tremor of muscles, enlargement of lymphatic

glands, and anæmia. Local sweating, particularly on the plantar aspects of the hands and feet, which are cold, is often distressing. The disease may subside or pass into the chronic form.

In the chronic form, which is much the commoner, the onset is gradual and insidious, and the progress much slower. In about two-thirds of the cases the small joints of the hands are first affected, in others, those of the feet and the larger joints—wrists, elbows, knees, &c.—become gradually included. There is the same symmetry. The temporo-maxillary articulation is often early involved. This happens more frequently in arthritis deformans than in any other joint trouble, the next in order being gonorrhœal rheumatism. Pain is very variable, sometimes absent, sometimes very great, particularly at night, being generally increased by movement, especially when prolonged, and by warmth. It is generally very fitful. The disease tends to steadily progress and produces gradually the changes already described under morbid anatomy; but at times it subsides, even in its early stages, and may remain quiescent for long periods, even years. Alternate quiescences and exacerbations are very common. There is a tendency to deflection of the fingers, the terminal phalanges deviating to the radial side and the others and the wrists to the ulnar side. The muscles atrophy, the skin becomes glossy and shows pigmented (burnt sienna colour), irregular patches, and subcutaneous fibrous nodules occasionally appear. There is no tendency to cardiac lesions. A great number of the cases, especially in elderly people (perhaps the majority), finally reach a permanently quiescent stage in which the general health is excellent.

Diagnosis.—Easy in well-marked cases, but may be difficult in the acute or in single joint cases. A careful consideration of the symptoms and the course of the case will help to clear it up (*vide* Table, page 326).

Prognosis.—Good so far as the immediate mortality is concerned, but the acute form of the disease is very hopeless and must run its course. In the chronic form much good may be done if an early diagnosis be made.

Treatment.—In its worst forms the treatment of this disease is very hopeless save for the relief of pain, and in all cases its success is largely dependent on the early recognition of the nature of the ailment. The patient must be well fed, warmly clad, and if possible sent to Algeria, Egypt, or a similar warm and dry climate during the winter months. The food should include plenty of butcher meat and the maximum of nourishment the patient can assimilate. Wines, especially port and burgundy, are helpful. Internally we may order cod oil and malt extract from the point of view of nutrition rather than medication. Salicylates relieve pain in certain cases and should be tried until they fail. Iron, and especially the iodide of iron, and certainly arsenic are of great value. Potassium iodide is often useful, and Luff recommends guaiacol carbonate (in 10 to 20 grain doses) and also methylene blue (in 2 grain tabloids twice, and later thrice, daily), but prefers the former, which he thinks may antagonise the supposed toxin if the disease is really due to a specific micro-organism. Locally, massage tends to arrest muscular wasting and prevents ankylosis, besides often aiding in the removal of infiltrations. Counter-irritation in all the forms referred to under rheumatic arthritis may be tried, but iodine is of very special value in some cases. Baths are often most helpful, especially the radiant heat, electric baths with alternating current, which should be begun early and persisted in for a long time, and the peat bath (Strathpeffer), and hot water douches. Lately the high frequency currents have been found to greatly relieve pain, and when used for a long

time permanent improvement may be anticipated. Occasionally excision has been practised with success for ankylosis. It is only requisite to add that when Heberden's nodes are the only or chief manifestation of this disease little treatment beyond good food is required.

RICKETS.

Lat. *Rachitis*. Fr. *Rachitisme*. Ger. *Rhachitis*. Syn. *Morbus Anglicus*, *Englische Krankheit*.

DEFINITION—A constitutional disease of early childhood, associated with impaired nutrition of the whole body, manifested by deformities of the bones and weakness of the ligaments and muscles.

Etiology.—Age is a most important factor, for the great majority of cases occur during the period of primary dentition (from the sixth to the twelfth month of life). It is, however, not uncommon for it to begin later, from the first to the second year, but very rarely still later; while it is also comparatively rarely seen before the sixth month. The sexes are equally affected, though some statistics give a somewhat greater incidence in males. The nature of the food is undoubtedly the most potent factor in the causation of the disease. It is most commonly by far met with amongst hand-fed infants. There is some dubiety about the relative importance of particular foods, but all are agreed that the quality is of more importance than the quantity. The balance of evidence, both in children and the lower animals, seems to place fat first in influence, proteids next, and lime salts closely after. Artificial foods and diets generally rich in carbohydrates but poor in fats, proteids and lime salts are most provocative of rickets. But diet is not alone to blame, for the disease may show itself when the

diet is everything that it ought to be. Unhealthy surroundings, want of fresh air and sunlight, cold and damp have an important influence, probably by impairing the digestive powers and the general vitality, hence the disease is most prevalent amongst the poor of large and crowded cities. The disease is thus essentially due to disordered nutrition, brought about mainly by unsuitable food but also by impairment of digestive powers, caused in most cases by a combination of the factors above mentioned. It is important to note that the bones are not the only tissues which suffer, for all the other tissues in the body participate, particularly the ligaments, muscles and certain viscera, and the prominence of the bone changes is due to their being more profoundly affected, mainly because they are at a period of great physiological activity.

Morbid Anatomy.—I. CHANGES IN THE BONES.—The normal physiological changes which subserve bone formation in both cartilage and periosteum become deranged.

(1) *In the epiphysis* the cartilage cells multiply irregularly and more abundantly, and lose their typical arrangement in longitudinal rows. Medullary spaces form irregularly, many appearing in the midst of the cartilage, often far above the usual line of bone formation between the shaft and epiphysis. Calcification of the cartilage takes place very irregularly. Ossification, resulting in spongy bone formation, is also very irregular. Thus islands of spongy bone or calcified matrix may be found in the cartilage above the epiphyseal line, and islands of unaltered cartilage below it.

(2) *In the periosteum.*—The changes are most marked in the neighbourhood of the epiphysis, but may extend all along the shaft. The osteogenetic layer of the periosteum is greatly increased, and much more vascular than normal, and an unusual amount of spongy, very vascular bone or simply calcified matrix is formed, which microscopically may present the appearance of a layer of blood-clot between

the surface periosteum and the bone. The actual result of the processes, therefore, both in epiphysis and shafts, is the over-production of a soft spongy tissue, which takes up more room than normal and cannot stand strain in the same way, hence there is thickening of the ends of the long bones and bending of their shafts. The percentage of lime salts may be reduced from about 65 to 50 or less, sometimes falling to 32 or even lower. The flat bones of the skull, &c., being membrane bones, show much the same periosteal changes, resulting in irregular thinnings and thickenings, and even alterations in shape when subject to strain, as in the bones of the pelvis. All these changes may largely disappear when the processes subside, and even a considerable amount of deformity may straighten itself out. In other cases, however, they give rise to a sclerotic condition, especially upon the surface, and the deformities become permanent. These changes may be seen in varying degrees of intensity in all the bones of the body. The skull bones may show patches of great thinness (chiefly from absorption of the inner table), often called *craniotabes*, similar to that of inherited syphilis, or thickened bosses over their protuberances, especially frontal and parietal, giving the large square forehead and flat head so often seen. The fontanelles remain unduly open. The teeth appear later and decay early. In the spine the normal curves are replaced by a single one convex backwards, commencing near the first dorsal and ending at the sacrum. It is believed to be due mainly to muscular weakness. The transverse diameter of the pelvis is increased and the antero-posterior diminished, giving a kidney-shaped cavity, which later in life may cause difficulty in parturition. The thorax shows beading of the ribs and prominence of the sternum. The beading of the ribs is one of the earliest and most characteristic signs of rickets. It is due to a thickening of the ends of the ribs at their junctions with the cartilages. It is symmetrical and generally most marked below. It is easily palpable, and is

known as the "rickety rosary." It is to be noted that it occurs all round the circumference and not merely on the outer side. In addition, the ribs are often flatter than normal at the sides, and the sternum projected forwards in a rounded, not a sharply-ridged fashion. This constitutes the "pigeon breast" of rickets.

In the limbs there is thickening of the ends of the long bones, particularly of their epiphyseal extremities. This is well seen at the wrists and ankles, but it occurs at the other joints as well. The shafts of the bones may be thickened, but they are bent only in a minority of cases, and if care be taken this may be avoided altogether. The bendings are generally exaggerations of the normal curves. Bow legs is the commonest form in the lower limbs, but knock-knee also occurs.

2. CHANGES IN SOFT TISSUES.—The muscles are thin and badly developed and flabby. The ligaments are easily stretched and thus become lax and the joints loose. The blood generally shows a distinct anæmia. The liver is often enlarged and may show cirrhosis. The spleen is occasionally also enlarged. The abdomen is prominent ("big belly"), and this is due to the weakness of the abdominal muscles and the distension of the intestines with flatus, and sometimes also the enlargement of the liver and spleen. The lungs show anterior bands of collapse, accompanying emphysema, and are especially prone to bronchitis and pneumonia.

Symptoms.—The disease has a very gradual onset. There may be signs of respiratory or gastro-intestinal catarrh, such as bronchitis, *laryngismus stridulus*, vomiting and diarrhoea. There is no fever, but the child is restless, especially when asleep, and is apt to toss the bedclothes off. He also often perspires, particularly about head and brow, when asleep, and is peevish and irritable. The changes in the bones, above described, gradually show

themselves. Diarrhoea becomes frequent, and at times alternates with constipation. The stools are pale and very offensive. Fever may appear, but is evanescent unless due to some complication. The child is often mentally backward but sometimes precocious and sharp. In addition to laryngismus stridulus, which is very common (probably characteristic of rickets), there may be tetany or general convulsions. There is sometimes a peculiar rolling of the head and nystagmus.

Diagnosis.—From inherited syphilis, with which Parrot confused it, by the later appearance of the symptoms, by the history, &c. From hydrocephalus by the changes in the chest and limbs. A certain degree of hydrocephalus is often combined with rickets.

Prognosis.—Very favourable. It is only fatal through some complication, but permanent deformities may be produced.

Treatment.—The great secret of success in treating rickets is to give proper diet and to study that the sufferer has fresh air, warm and sufficient clothing and hygienic surroundings. Want of fatty matters in the food (poor milk, if the child has been or is still being suckled) indicates the proper plan of treatment. Cream, butter or cod-liver oil are invaluable, and in many cases raw meat juice or scraped raw meat prepared as a sandwich should be given. It is hardly necessary to add that in certain cases the digestive powers require assistance, at least for a time, and the motions should be carefully watched so as to give an early indication of insufficient digestion or fermentation. In most cases the patients must be kept as much as possible from walking or creeping about until the tendency to bending of the long bones is out-grown, and various means have to be adopted to keep the little ones as much as possible in the open air

and yet "off their legs." Salt water bathing and sea air are most beneficial. Phosphorus, which may be administered in the form of phosphates, and syrup of the iodide of iron are good tonics. A little sensible advice to the mother of a rachitic child may do much to save the development of future cases.

GOUT.

DEFINITION—A constitutional disease characterised by deposition of salts of uric acid combined with soda in and around the joints, along with a certain amount of inflammation in the affected joints.

Etiology.—There are certain conditions which predispose to gout more or less strongly. It affects men more than women, and in both it comes on in middle or advanced life. Heredity is a most important factor, a gouty diathesis descending from generation to generation. Habits of life have also a marked influence. It is particularly apt to show itself in those addicted to high living; over-indulgence in alcohol, in the form of rich wines and malt liquors, being particularly provocative. An attack of gout frequently follows upon some injudicious dietary indulgence, or violent exercise, or an injury, or worry, or fit of anger, or other violent emotions, but none of these are in themselves sufficient to cause gout. The real exciting cause is related in some way to uric acid, though the exact relationship is still undetermined. Free uric acid does not exist in the blood. It is combined with soda as a quadriurate, which is a comparatively unstable salt, and is easily converted into the biurate, which, being much less soluble, is deposited in the tissues. Why the cartilage and fibrous tissues of the joints, and only of certain joints, are selected we do not know, but it may be connected with the comparatively poor

arterial supply of those tissues and the greater strain that is thrown upon them. Many regard gout as a neurosis, a slight disorder of certain nerve centres, leading to derangement of the joint nutrition, and thus determining the urate infiltration. Since the kidney is diseased in most, if not all, of the cases of gout, this organ is held by many to be responsible for the disturbance which occurs in the secretion of urates. The amount of uric acid excreted daily in health is about 8 grains, and from careful analyses it would seem that this amount is greatly diminished for a variable period preceding an attack of gout, rising to normal again during the attack—the so-called uric acid showers. This failure to excrete the proper amount of uric acid causes a steadily increasing excess (as quadriurate of soda) to circulate in the blood. Others, again, ascribe the increased uric acid of the blood to the nucleins produced by the destruction of leucocytes, the amount depending upon the degree of leucocytosis present. This has, however, been shown not to be a constant feature in some other diseases with marked leucocytosis, and consequently we cannot yet be said to have solved the problem of the relation of uric acid to the causation of gout.

Morbid Anatomy.—The metatarso-phalangeal joint of the great toe, right oftener than left, is most frequently affected. The other small joints of the feet or hands may be subsequently or simultaneously affected. The larger joints—wrist, elbow, knee, &c.—may also suffer. The disease begins in the centre of the cartilage in the form of whitish-grey, chalky-looking patches or continuous deposit, looking as if it had been powdered with a white substance like plaster of Paris, or daubed with a white paint, but on looking more closely the deposit is seen to lie in the substance of the cartilage below the surface, the covering epithelium being intact. There is no histological evidence of any degenerative or other change in the cartilage prior to the

deposition of the white substance. This substance is entirely composed of biurate of soda in the form of fine needle-like crystals aggregated into variously-shaped masses. At the same time, or soon after, there is a similar deposit patchily distributed in the lateral or inter-articular ligaments. The deposit gradually increases until the whole extent of the cartilage and the ligaments of the joint are thickly infiltrated. All the periarticular fibrous tissues, including the periosteum, suffer, but there is no deposit in the bone. The deposit causes an inflammation within the cartilage, synovial membrane, and other fibrous tissues of the joint, and the rapidity with which it occurs and its copiousness largely determines the amount and sharpness of the consequent inflammation with its attendant pain and swelling, due to effusion into the joint and œdema of its peri-articular tissues. The effusion is clear, and though it may show urate crystals it does not suppurate. The results of the inflammation are somewhat different at the periphery and centre of the joint. In the latter situation it soon leads to necrosis of the cartilage, with subsequent erosion, so that the uratic deposit now bounds the cavity of the joint (a true incrustation), into which some of it may be displaced. At the periphery, on the other hand, the proliferative and formative results are more marked, as in arthritis deformans, probably because of the greater vascularity and lessened pressure. The synovial fringes and margins of the cartilage thus become thickened, and bone formation may take place therein, causing various nodosities and lippings of the ends of the bones. This alteration along with the uratic infiltration are the chief explanations of the joint deformities, which are often marked in long-standing cases — “chronic deforming gout” as it is often called. The whole joint may be disorganised, and dislocation may result in severe cases. The fingers may become deflected to the ulnar side. The inflammation spreads to the bone, causing some absorption of the cancellous tissue. It may extend to the neighbouring

tendons, their sheaths, and the subcutaneous tissues. Uratic deposits gradually accumulate therein and may assume a large size, causing the joint to appear nodular and rounded, and the skin over it to be tensely stretched and thinned, thus clearly revealing the white deposit underneath. These deposits are called tophi, and the skin over them sometimes gives way, allowing them to escape *en masse* or gradually as a thick creamy-looking substance. They occur most frequently in the ears, and in the skin and bursæ over or near joints.

Clinical Varieties.—(1) Regular, where the joints are affected; (2) irregular, where other situations are affected; (3) retrocedent or metastatic, where the attack in a joint suddenly subsides and appears in some internal organ.

Symptoms.—1. REGULAR GOUT.—This is oftener spoken of as acute or chronic. Others look upon gout as always a chronic disease subject to acute exacerbations. A typical attack comes on acutely with little or no warning, the patient being awakened, generally in the early morning, by pain in the affected joint (mostly the great toe). The pain is very severe and quickly increases, becoming excruciating. The joint is swollen and the skin over it tense and red, and showing distended veins. The temperature rises (100 to 102°F.) and the pulse is quickened. The mental condition is clear, but there is great irritability and restlessness. After a few hours a gentle perspiration comes on and the pain abates considerably, the patient generally falling into slumber, from which he awakes in comparative comfort, which continues throughout the day, though the joint is still swollen, shiny, red and tender. The urine is scanty and high-coloured. There is generally thirst, anorexia, and constipation. During the succeeding night, and probably for one or two and sometimes more thereafter, he may have recurrent attacks, each generally diminishing in severity, and after a week or

two he is completely restored to health, often apparently a heightened health. He may never have another attack, but this is exceptional, for usually after a varying interval—a week, months, or years—further outbreaks follow, involving generally a greater number of joints each time, and becoming more prolonged, until the condition of chronic gout is established, in which the joints show the thickenings and deformities described under morbid anatomy. The urine generally shows the features characteristic of granular contracted kidney, and there is often some glycosuria. The same arterio-capillary sclerosis and cardiac hypertrophy are also present.

2. IRREGULAR GOUT.—It may occur either in combination with regular gout or without it, and the latter is more frequent. All its forms are probably due to the deposit of biurate of soda in the tissues—*e.g.*, the valves of the heart, walls of vessels, vocal cords, cerebral and spinal meninges, &c.—causing many different forms of catarrh, such as pharyngitis, laryngitis, bronchitis, neuritis, urethritis, iritis, eczema, and so on.

3. RETROCEDENT OR METASTATIC GOUT.—It may follow upon the sudden application of cold. Its causation is obscure. It may be due to deposition of biurate of soda or to a reflex nervous hyperæmia. It may attack the stomach, causing pain, vomiting, &c.; or the heart, causing pain, dyspnœa, syncope, &c.; or the brain, causing apoplexy, congestion, headache, delirium, &c.

Diagnosis.—Acute and chronic rheumatism, arthritis deformans, pyæmic arthritis, gonorrhœal arthritis, and traumatic arthritis are the affections most likely to be confused with gout. Certain distinctions have already been drawn between them, and the three most important of them are further differentiated in the Table, page 326.

Garrod's thread test should also be used. Take two drachms of the serum, or the fluid raised by a blister

applied at some distance from the inflamed joint, and place them in a large watch glass, and then acidulate with acetic acid. Place a piece of thread in the prepared fluid, and let it remain for some time. Examine the thread with the microscope for crystals of uric acid, and confirm by adding nitric acid and ammonia to form the purple colour—murexide.

Prognosis.—It is favourable in most uncomplicated cases, and under appropriate treatment it does not tend to materially shorten life. If the attacks become frequent, or the disease affects the heart or other important organ, especially in the form of irregular or retrocedent gout, the outlook is much less favourable.

Treatment.—The gouty patient does not merely want relief from pain, but his diet, mode of life and amount and manner of exercise all require to be regulated, and those who may have ancestral claims to a gouty constitution will do well to anticipate the first onslaught of the enemy, at least when they have reached the age of forty.

DIET.—During an attack of acute gout, milk and milk puddings should constitute the staple diet. Authorities are much at variance as to the desirability of allowing soups, beef-tea and so forth. Certainly it is better if the patient can take milk to limit the diet as nearly as possible to that during the acute stage. Between the attacks, do not give an excess of carbohydrates, nor, on the other hand, give nitrogenous diet alone. Meat, which includes white meat and fish, should not be taken more than once a day in most cases, and sugar should be replaced with saccharin or saxin. Bread and vegetables, especially the green varieties, may be eaten freely; while fruit, both fresh and stewed, is admissible and excellent. Amongst articles of food which should be taken at least in great moderation by gouty patients are—shell-fish, green peas and asparagus,

rich pastry and cheese. Roberts considers common salt, and in fact all soda salts, to be injurious, because they tend to unite with the quadriurate to form a very insoluble biurate of soda in the joints and tissues, and he therefore prohibits salt meat and fish, the free use of common salt, and all sodium salts in purgative or other medicines. He advises the patient to take potassium chloride as a substitute for common salt, although its taste is extremely unpleasant. A moderate amount of fat and butter is not prejudicial. All so-called fruity wines are prohibited, and especially port and burgundy; sweet champagnes, beer, porter, and cyder, should on no account be permitted. Some physicians have begun to allow dry cyder, bitter beer, and other previously forbidden liquors, but it is better to err on the safe side. Whisky, in very small quantity, and some of the Moselle wines are among the best alcoholic beverages, but all alcohol must be very carefully limited in amount.

MEDICINAL REMEDIES (Local and Internal).—In an acute attack the affected joint or joints (so frequently the metatarso-phalangeal joint of the great toe) should be fomented, if necessary, with opium fomentations, raised on a gout stool, and wrapped, after or between fomentations, in cotton wool. Cold applications should never be used, and on no account should the lancet or leeching be thought of. At the same time the patient may be given a brisk purge, by preference calomel or blue pill, followed next morning by a saline cathartic, such as magnesium sulphate. With the least possible delay he should also be given 10 to 30 minims of tincture of colchicum seeds or colchicum wine, and the dose repeated every four hours till the pain abates. This may be combined with citrate of potash or lithia in 10 to 30 grain doses. The colchicum must be carefully watched, and care taken to satisfy oneself that the kidneys are discharging their functions. Probably both the citrates of potash and lithia, whichever is selected, act

more as diuretics than in any other way. Piperazine in 15 to 20 grain doses is a supposed solvent of uric acid, but its beneficial action in this respect is very dubious.

In a more chronic case, potassium iodide, guaiac, salicylate of soda, and quinine are useful remedies; and the hot air bath locally to the joint or limb is in many instances of undoubted value. Where irregular or retrocedent gout is present, colchicum is useful, and should be given; but cardiac tonics, gastric sedatives and so forth must also be administered, according to the symptoms present. In all these cases the utmost attention should be paid to the question of diet. Baths are certainly to be commended. For one thing, large quantities of water are drunk, free purgation is obtained as well as free diuresis, and regular and limited meals replace a dietary often injudicious in the extreme. Nearly all spas, home and continental alike, cater for gouty patients, and it is only necessary to mention Bath, Buxton, Strathpeffer, Harrogate, Carlsbad, Aix-les-Bains, and Tarasp, among a long list of suitable baths.

Lastly, the gouty subject should be enjoined to take regular exercise, to attend to the hygiene of the skin by daily cold baths, and occasionally to indulge in a Turkish bath if the heart and circulation permit. Chills must, on the other hand, be avoided, and a sufficiency of warm clothing should be worn by every one who has any susceptibility to gout.

DIFFERENTIAL DIAGNOSIS OF THE FOLLOWING DISEASES.

(After GARROD.)

GOUT.	RHEUMATISM.	RHEUMATOID ARTHRITIS.
1. Strongly hereditary.	1. Less so than in gout.	1. Less so than in gout.
2. Much more frequent in males.	2. More frequent in females up to 15, afterwards more frequent in males.	2. More frequent in females.
3. Seldom occurs before puberty; generally much later.	3. More frequent in the young before middle age.	3. Occurs in both young and old.
4. Induced by high living, wine and malt liquors.	4. Occurs in the weak, and not caused by wine, &c.; excited by cold and damp.	4. Often induced by depressing causes and sometimes excited by cold.
5. One or more of the smaller joints particularly affected in early attacks, and especially the great toe and joints of foot below ankle.	5. Large joints more affected than small, usually several in number, and very rarely those of foot except ankle.	5. Large and small about equally affected.
6. Great pain, œdema, and desquamation of cuticle.	6. Pain less intense; seldom œdema.	6. Less pain, much swelling, and often some œdema.
7. Does not usually induce acute inflammation of structures of the heart.	7. Often causes acute pericarditis and endocarditis.	7. No tendency to cause disease of the heart
8. Febrile disturbance moderate.	8. Febrile disturbance great, more than from local inflammation.	8. Little febrile disturbance.
9. Paroxysm periodic in early attack.	9. The attacks not periodic.	9. No periodicity.
10. Early attack lasting a week or ten days.	10. Attacks generally much longer.	10. Duration of attack indefinite.
11. Blood rich in biurate of soda	11. No biurate of soda in blood.	11. No biurate of soda in blood.
12. Constant deposit of biurate of soda in inflamed cartilage and ligaments.	12. No deposit of biurate of soda.	12. No deposit of biurate of soda.
13. Often associated with kidney disease.	13. Not so associated.	13. Not so associated.
14. Often produces chalk stones externally (tophi).	14. Never causes chalk stones.	14. No chalk stones produced, but swelling of the joints.

DIABETES MELLITUS.

Lat. *Diabetes, Diabetes mellitus.* Fr. *Diabète, Diabète sucré.*

Ger. *Diabetes, Zuckerharnruhr.*

DEFINITION.—A disease characterised by an increased discharge of pale urine of high specific gravity, *persistently* impregnated with sugar, accompanied sooner or later with great emaciation.

Etiology.—Diabetes mellitus occurs at all ages, but is most frequent between middle and older life. It is commoner in men than women, and among the Jews and Hindoos than other races. Heredity is of considerable influence, and so is nervous strain, shock and worry, and a sedentary life. Obesity, gout and syphilis are supposed to predispose to it. It is difficult to say what are its exciting causes. There is sometimes a history of its following quickly upon some severe emotion, or cold, or head or other injury, but in many cases there is apparently no exciting cause.

Pathogenesis.—1. THE NERVOUS THEORY.—Since Claude Bernard's classical experiment showing that glycosuria followed upon puncture of the floor of the fourth ventricle, evidence of a lesion there has been carefully sought for, but found in very few cases, and histologically the evidence supporting a nervous origin of the disease is very slight, there being no detectable structural change to be made out. On the other hand, there is considerable clinical evidence connecting the disease with nervous shocks and disturbances, and its incidence is greater among brain workers than among manual workers, so that it is at least possible that certain nervous disorders may act as exciting causes.

2. THE HEPATIC THEORY.—The nature of glycogenesis is still imperfectly solved by physiologists, and we are hardly likely to understand fully the relation of the liver to glycosuria until the physiological problem is elucidated. The view most widely accepted is that the sugar and proteids of the food taken to the liver by the portal vein are transformed by the hepatic cells into glycogen and reconverted as required into sugar, which leaves the liver by the hepatic veins. This sugar circulates in the blood as dextrose, and is destroyed in the muscles and the tissues. Its amount in health (0.12 per cent.) is kept constant by a correlation between sugar formation in the liver and its destruction in the tissues. Another view, supported by Pavy, holds that the sugar of the food is converted by the liver and intestinal villi into fat, and its appearance in the blood is due to the liver not destroying it all, but allowing a little to pass on into the hepatic vein. According to the first view, an abnormal glycosuria may arise, either from an increased production of sugar in the liver or its diminished destruction in the tissues. According to the second view, it is due to an imperfect destruction of the sugar by the liver. There is an abnormal amount of sugar in the blood (hyperglycæmia) in diabetes, and in experimentally produced hyperglycæmia the amount of glycogen in the liver is diminished. This is in keeping with both the theory of increased formation of sugar by the liver and that of imperfect formation by that organ. It may be, also, that at least in some cases of diabetes there is decreased destruction of sugar in the tissues, for glycogen is formed in animals fed on a proteid diet, and in severe diabetes the glycosuria continues though the diet be a purely proteid one, the proteid molecule giving rise to urea, CO_2 , water and a carbohydrate. The increased excretion of urea favours this view; but, on the other hand, the fact that no increased excretion of CO_2 has yet been demonstrated is against it.

3. THE PANCREATIC THEORY.—The pancreas has been found to be variously diseased in a large number of cases of diabetes, and complete extirpation of this organ in dogs causes diabetes. It is supposed that the pancreas has two secretions—its intestinal secretion, which has no influence in this way, and an internal secretion which, passing into the blood, either checks sugar formation or favours its destruction, and if the disease of the pancreas be so extensive or of such a nature as to destroy this internal secretion diabetes is the result.

4. THE MICROBIC THEORY.—The evidence in its favour is very slender, and depends chiefly upon experiments showing that dogs may show glycosuria after the injection of certain pyogenic microbes into the pancreatic duct, and on the clinical examples of diabetes occurring in both husband and wife apparently pointing to contagion. Even if all these theories be accepted, it is not possible to explain all cases of diabetes by their means, so that we are forced to conclude that the disease, in all probability, may arise from several different causes, either alone or in combination, at present imperfectly known.

Morbid Anatomy.—Although the changes found in the organs and tissues of the body are considerable, they are mainly referable to the nutritional failure, and can hardly be regarded as characteristic. The nervous system may show a tumor, colloid masses, sclerosis or other change in the medulla, congestion, oedema, or cyst-like formation elsewhere in the brain, or some change in the cord, but there is no constant lesion. The heart is pale and sometimes hypertrophied or fatty. The blood is usually normal, but sometimes shows fat as fine drops, which run together after death and give the appearance of fat emboli. It contains an excess of sugar, sometimes as high as 0.4 per cent. It may be detected during life by a simple staining method, which will occasionally prove an aid to diagnosis. A dried

blood film is fixed and then stained by a 1 per cent. solution of either Congo red or methylene blue. With the former the blood-corpuscles of diabetic blood remain almost colourless, instead of becoming bright red as they do in health; with the latter a faint greenish-yellow instead of blue. The lungs show important secondary changes, the most frequent and important, after mere congestion and œdema, being tuberculosis and (less frequently) pneumonia or gangrene. The liver is usually enlarged and sometimes fatty. Occasionally it presents a gross lesion, particularly a cirrhosis. This cirrhosis is sometimes associated with bronzing of the skin, the *Diabète Bronzé* of Hanot, which he regards as a distinct disease. The pancreas often shows changes which have already been referred to. The stomach is often dilated. The kidney changes are always secondary, enlargement and slight fatty change being most common.

THE URINE.—The quantity is greatly increased (sometimes reaching about twenty pints), except in mild cases, when it may be as low as normal; the colour is pale and the specific gravity high (average 1030 to 1040), though in mild cases very little if at all above the normal. The urea, kreatinin, ammonia, phosphates, sulphates and chlorides are in excess. The amount of sugar (dextrose) varies from one to twelve per cent., or two to twenty-four ounces per diem. In severe cases it continues though carbohydrates be excluded from the diet, but in others it disappears. In some cases it disappears for a time and then returns.

TESTS FOR SUGAR.

1. QUALITATIVE.—(1) *Fehling's Test*.—This is the one most commonly used. About a drachm of the Fehling's solution is first boiled in a test-tube. This tests alike the purity of the solution and the cleanliness of the test-tube. Now add the urine drop by drop. A yellowish or brick-red precipitate indicates sugar. If there be no precipitate add more urine until it equals the solution in bulk, then boil for at least half-a-minute and set aside. If a copious typical precipitate forms

quickly there is no doubt as to the presence of sugar; but a scanty, slowly-formed and not quite typically coloured precipitate is not so trustworthy. It may be due to a small quantity of sugar, or to kreatinin, uric acid, lactose, glycuronic acid, and appear especially in the urine of patients taking salicylates, salol, chloral, chloroform, camphor, lactic or benzoic acids. A further test becomes necessary. Charcoal filtration is very useful here. Filter the urine five or six times through animal charcoal, which in most cases alters the reducing agents other than sugar, and a positive result is now much stronger.

(2) *Trommer's Test* is practically the same as Fehling's. Fill a test-tube half full of urine, add $\frac{1}{2}$ to $\frac{1}{2}$ a drachm of liquor potassæ and a few drops of a 1 per cent. solution of copper sulphate. Shake together and boil upper part of mixture. A yellow to brick-red precipitate indicates sugar. Both tests depend on the hydrated cupric oxide (CuOH_2O), which is kept in solution by the sugar in Trommer's and the tartrate in Fehling's solution, being reduced by heating in the presence of sugar to the hydrated cuprous oxide ($\text{Cu}_2\text{OH}_2\text{O}$).

(3) *Fermentation Test*.—This was the earliest devised test, and is still the best, though too tedious for ordinary use. Glucose is the only fermentable substance ever found in the urine. The urine if not distinctly acid should be made so with tartaric acid. It is well boiled for 10 minutes to exclude air. Fill an ordinary test-tube two-thirds full of mercury and then fill it up with urine and add a small piece of German yeast, previously well washed. Shake it up thoroughly, so as to form an emulsion free from lumps. Take a similar tube for control, to which no yeast is added. Invert them both in a bath of mercury and place them aside in a warm place for 12 hours. If sugar be present a bubble of gas will have formed at the end of the tube containing the yeast. The test can also be made quantitative by comparing the densities of the two specimens. The amount of sugar is got by reckoning each degree of density lost as 1 grain of sugar per oz. Two similar-sized, carefully-corked bottles are sometimes used to contain the urine, but the result is less reliable. Two Southall's ureometers may also be used, instead of test-tubes and a mercury bath, by filling the long limb completely with the urine.

(4) *The Phenyl-hydrazin Test (Von Jaksch)*.—Place in a test-tube about half-an-inch (or "twice as much as will lie on the point of the blade of a knife") of powdered phenyl-hydrazin hydrochloride and a little more of powdered acetate of potassium or soda, and then add 3 or 4 drachms of urine. Heat for two minutes without shaking and if the salts do not dissolve add a little water, and place the test-tube in boiling water for 20 to 30 minutes. Allow it to cool (stand it in cold water), and a yellow deposit is formed if sugar is present, which,

under the microscope, is seen to consist of radiating clusters of needle-shaped crystals of phenyl-glucosazone.

There are many other tests, such as Moore's, the picric acid test, &c., none of which are so good or reliable as those given above.

2. QUANTITATIVE.—(1) *Fermentative Method* (page 331).

(2) *Fehling's Method*.—The solution (Fehling's) is of such a strength that 10 c.c. will take '05 gramme of sugar to completely reduce all its cupric sulphate into cuprous oxide. Mix the whole 24 hours urine and take 5 c.c. thereof, add 95 c.c. of distilled water and mix together so that the urine is diluted 20 times. Fill a burette with this diluted urine. Place 10 c.c. of the Fehling's solution into a white porcelain dish and dilute it with 40 c.c. of distilled water and boil. Turn the stop-cock of the burette and allow at first 4 or 5 c.c. of the diluted urine to flow into the boiling Fehling. Stir with a glass rod. Continue to add more diluted urine, stirring carefully each time until all the blue colour of the Fehling is discharged and the cuprous oxide is precipitated. Near the end of the process add the Fehling carefully drop by drop, and take away the bunsen occasionally and allow the cuprous oxide to settle in order to estimate better the colour of the fluid. Whenever the colour is completely discharged read off the number of c.c. of diluted urine used, say 40 c.c., this will equal '05 gramme of sugar, therefore $\frac{.05 \times 100}{40}$ represents the percentage, but the urine was diluted 20 times, therefore $\frac{.05 \times 100 \times 20}{40} = 2.5$ is the percentage of sugar present in the undiluted urine. Again, 40 c.c. of the urine diluted 20 times contain '05 gramme of sugar, therefore 2 c.c. of undiluted urine contain the same. Suppose the total number of c.c. passed in the 24 hours = 2000 c.c., then $\frac{.05 \times 2000}{2} = 50$ grammes, the total amount of sugar excreted in the 24 hours.

(3) *Pavy's Method*.—Pavy's solution contains ammonia, which keeps the cuprous oxide in solution, and it is therefore easier to judge of the colour of the solution. The solution is only one-tenth as strong in copper as Fehling's, hence 10 c.c. = only '005 gramme of sugar, and since the ammonia is volatile, it is necessary to boil the solution in a beaker whose neck is loosely plugged with cotton wool, through which passes a tube connected with the burette. Otherwise the steps are the same.

Other substances occasionally found are—(1) Albumen, which should be got rid of by heat and filtration before

testing for sugar; (2) diacetic acid, found frequently in severer forms of the disease, and tested for by adding liq. fer. pechlor. drop by drop to the urine until a clear, deep Burgundy-red solution appears; (3) acetone, of like significance, and tested for by adding a few drops of freshly made concentrated nitro-prusside of sodium to 5 or 6 c.c. of the urine rendered alkaline by the addition of liq. potassæ. A red colour appears and soon disappears. A little acetic acid is added and a deep red or violet colour will be produced if acetone be present. (4) β -oxybutyric acid, occasionally found only in severer forms and considered by Von Noorden to be the sole cause of diabetic coma.

Symptoms.—Two types—a severe and a mild—are recognised. They differ from one another mainly in the former generally showing itself in younger people, in running a more acute and rapid course, in being attended by much and rapidly occurring emaciation, and in a greater intensity of the glycosuria, polyuria, and thirst. The disease usually sets in insidiously, the patient complaining of frequent micturition, unusual thirst, or of weakness and wasting. The frequent micturition, although less severe at night, interferes with sleep, and thus causes constitutional depression. The thirst becomes intense and distressing, and the amount of water drunk explains the polyuria. It is greatest an hour or two after meals. Occasionally it is not troublesome, and then the polyuria is slight. The appetite is often voracious, but in spite of the large food consumption there is steady loss of weight. A considerable amount of subcutaneous fat may be present, particularly in the early stages of the disease in older people, who may show no emaciation for years. There is often a persistent sweet taste in the mouth, and the breath may have a sweet odour. The tongue and lips are dry and red, the teeth often decay and fall out, and constipation is usual. The skin and hair are dry and harsh. Sexual desire is generally lost, and in women menstruation is

generally deficient or absent. The temperature is usually subnormal, but occasionally it is raised at the onset of acute symptoms. The pulse is tense at first, but tends to become small and feeble in the later stages. It is not quickened except some complication is about to come on, a rapid pulse being one of the early signs of the onset of coma. Coma is a very grave condition. It is responsible for about half of the deaths from diabetes. It may come on suddenly without any apparent reason, or supervene upon fatigue or some sudden change of diet. Various symptoms may precede an attack. There may be some nausea, vomiting, or other gastric disturbance, or headache and difficult sighing-like breathing ("air hunger"), or the pulse becomes rapid and weak, and the skin cold. The urine often diminishes in quantity, and its sugar contents often lessen. The patient then becomes apathetic and drowsy, and later falls into a deep and fatal coma. Death supervenes in a few hours to five days. The general resemblance to uræmia suggests a failure in renal function or the presence of some toxic agent in the blood. Acetone and diacetic acid have been blamed, also β -oxybutyric acid, and, with still greater probability, according to others, β -amidobutyric acid is a still more likely cause. The characters of diabetic urine have already been given. The presence of sugar is its cardinal feature. In severe cases it persists, even though carbohydrates be excluded from the diet, but in mild cases it may be largely or completely banished. This raises the question of the possibility of a *non-diabetic* or *temporary glycosuria*. There is an *alimentary glycosuria*, in which, after the excessive ingestion of sugar, but not starches, sugar is found in the urine. It is always small in amount, usually under 5 per cent. It indicates an imperfect power of assimilating sugar, but its relation to diabetes is still an open question. It may signify a tendency towards diabetes, but it has been seen in patients for years, in whom no other sign

of ill-health has followed Glycosuria is also a common associate of gout, of many surgical injuries, some nervous diseases, some specific fevers, obesity, and other general maladies, in which its relationship to true diabetes is not quite certain.

Complications.—They are pretty numerous and referable to nearly all the systems of the body. The skin shows, frequently, boils and carbuncles, sometimes eczema, sometimes pruritus pudendi. Gangrene is not uncommon. The lungs show phthisis very frequently (in about a quarter of the cases in young people). The vessels frequently show arteriosclerosis. The nervous system often shows neuralgias, occasionally peripheral neuritis. There may be irritability, depression, melancholia or mania. Cataract is the most frequent complication in the eyes. It is of the soft variety, and occurs chiefly in the severe type in younger patients. Albuminuria is not uncommon, especially in the chronic cases.

Diagnosis.—Attention to the urine renders the diagnosis easy. *Vide* tests for sugar.

Prognosis.—Should the sugar persist in considerable amount and the weight and strength of the patient steadily and rapidly diminish, notwithstanding careful dieting and rest, the prognosis is very grave. Such cases in young people may prove fatal in a few weeks to months. Age is usually of the greatest importance in prognosis, as length of life is generally shorter the younger the patient. In mild cases, particularly those in older and obese people, the health may remain good for many years, but an acute stage may come on at any time. If the quantity of sugar and urea continue small, the weight and strength will generally be maintained, and hence particular attention should always be paid to the amount of urea present.

Treatment.—When the patient comes under observation we try, in the first instance, to find out the exact data of the case, *e.g.*, with regard to the amount of sugar and urea excreted, and the presence or absence of the derivatives of β -oxybutyric acid, to which at the present time we are inclined to attribute diabetic coma. It is only possible to estimate the amount of sugar after a period of careful dieting. For some days the sugar should be estimated while ordinary diet is being taken, then *gradually* carbohydrates should be eliminated, until the patient is taking a rigid “diabetic” diet. If the sugar disappears, the case is mild and might almost be termed functional glycosuria; if it does not, the amount still excreted is a guide as to the severity of the case. Generally it is possible, and even desirable, to allow some carbohydrates in the diet, and preferably bread in the form of toast, because a rigid exclusion of carbohydrates is believed to predispose to diabetic coma where it is at all imminent. The amount allowed, however, should not cause an appreciable increase in the sugar excreted above that passed upon the rigid nitrogenous diet.

DIET.—*The following are permissible*—Red and white meat (except liver), fish of all kinds (but excluding the shell-fish with large livers), soups, eggs, cheese, milk and cream, butter and edible fats of all kinds, green vegetables, mushrooms, fruits which are not too sweet, nuts (except chestnuts), the substitutes for bread—gluten, almond and cocoa-nut bread and biscuits, and certain forms of bran bread. We can allow water, alkaline mineral waters, tea and coffee (sweetened with saccharin), dry wines (such as dry sherry), Moselles, and unsweetened spirits in moderation.

The following are forbidden—Sugar and carbohydrates of all kinds, potatoes and other starchy vegetables, such as turnips, peas, beans, &c. All sweet fruits and fruit syrups, sweetened wines and spirits.

Above all things carefully arrange for as much variety in diet as possible, and especially where the appetite is capricious and the patient is losing flesh. The clothing should be warm, and soft flannel or silk should be worn next the skin. Exercise must be regulated. Generally an active out-of-door life, without permitting fatigue, and freedom from mental effort or business worries are enjoined. Cold bathing is good, and even a Turkish bath occasionally is most beneficial. Keep the bowels freely open: saline aperients being specially serviceable for this purpose. There are certain spas which are distinctly beneficial, notably Carlsbad, Marienbad, and Neuenahr.

MEDICINAL REMEDIES.—There are no remedies of striking value except the opium group and alkalies. Opium, morphia or codein in $\frac{1}{2}$ to $1\frac{1}{2}$ or 2 grain doses thrice daily often greatly diminishes the excretion of sugar, but such drugs produce constipation, and not infrequently severe dyspepsia. Alkaline salts, and especially sodium carbonate (100 to 200 grains per day), certainly have averted diabetic coma. Among other drugs, salicylate of soda in 20 to 30 grain doses thrice daily has proved of benefit in some cases, and nitro-glycerine, jambul, arsenic, strychnin and lactic acid are only a few of many remedies suggested, but which have not proved of much curative value. Lépine has tried a glycolytic extract, prepared either from the pancreas or from maltose, which he believes to have been of value in a few cases, but the statistics are unconvincing. The awful itching and eczema about the genitals are greatly alleviated by making the patient wash locally with boracic lotion after micturition. Diabetic coma is very often fatal, but in every case constipation, if present, should be at once relieved by an enema or other means, and an intravenous injection promptly administered of a 3 to 5% solution of sodium carbonate in normal saline solution (75% sodium chloride in distilled water). About two pints heated to blood temperature (100°F. or more) may be given, and the basili-

vein in the arm is generally selected. The result is nearly always a temporary improvement in the patient's condition. Sometimes sodium carbonate is given as an enema, but it is not nearly so efficacious. Always caution diabetic patients to avoid over-fatigue, mental strain, worry and constipation.

DIABETES INSIPIDUS.

DEFINITION.—A rare morbid condition, characterised by the excretion of a persistently excessive amount of urine of low specific gravity containing no sugar or other constant abnormal constituent.

Etiology.—It is fully twice as frequent in males as females, and though it may occur at any age, is most common in adult life. Heredity has a strong influence, a predisposition to the disease, if not the disease itself, being often transmitted. Tubercle and some acute febrile diseases seem also to predispose to it. As in Diabetes Mellitus, with which it has many points in common, cases have been frequently found to follow upon blows on the head, nervous shock, strain, &c. It is occasionally associated with some lesion in the floor of fourth ventricle, experimental puncture of which has also produced it. A nervous origin of its causation has thus considerable support, and a functional disorder affecting the vaso-motor system has been suggested among other things, but the problem is still unsolved.

Morbid Anatomy.—No constant gross lesion is found. The urine is clear and limpid, slightly acid, and very pale, with an average specific gravity of about 1005. Its outstanding feature is its excessive amount—eight or ten pints, but sometimes thirty to forty, per diem, roughly varying

with the quantity of fluid consumed. It contains no sugar, and its amount of urea is, as in health, directly dependent upon the diet.

Symptoms.—The chief symptoms are the great thirst, which gives rise to much distress if not satisfied; a dry skin, and, generally, emaciation sooner or later. Frequently the patient enjoys good health for years, but more commonly he becomes thin and debilitated and irritable, often complaining of lumbar pains. The appetite is good for a time, but fails in the later stages of the disease, when the progressive emaciation and loss of strength become marked.

Diagnosis.—From diabetes mellitus by the absence of sugar, and from chronic Bright's disease by absence of albumen and cardio-vascular phenomena.

Prognosis.—In children it is bad. In adults milder cases sometimes recover. Ordinary cases are generally very intractable, and though they may live a long time the ultimate outlook is very bad. When emaciation and loss of strength become pronounced, death is not far off. A fall in the amount of urea excreted to under 300 grains per diem is a serious symptom. It may be a forerunner of diabetes mellitus.

Treatment.—There are few diseases whose treatment is so unsatisfactory. Any causal agent should be sought for, and if discovered should be eliminated or antagonised. In our hands opium, ergot, nitro-glycerine and valerian have all been serviceable in different cases, and should be tried separately. Care to avoid ergotism is necessary. As a rule, it is not wise to limit the amount of fluid taken too suddenly or continuously. Galvanism in a few cases has proved of advantage.

Section 3.

DISEASES OF THE BLOOD, BLOOD-FORMING AND DUCTLESS GLANDS.

I.—*DISEASES OF THE BLOOD.*

(INTRODUCTION.)

There is a class of diseases which mainly affect the blood. Strictly speaking, every disease must affect the blood, but in other diseases the blood changes are either unknown or insignificant compared with the lesions produced in certain organs or tissues of the body, and *vice versa* even the so-called primary blood diseases must affect every other part of the body, but the changes produced therein are also either at present unknown or comparatively insignificant. The systematic examination of the blood as a regular clinical procedure in disease is now as necessary as that of the urine. Fresh films should first be examined, as they will often enable us to decide if a detailed examination is necessary. If the red corpuscles are of uniform size, well shaped, and of normal hæmoglobin content, we may safely infer that there is not much, if any, anæmia present, and it may not be necessary either to count their actual number or to estimate the amount of hæmoglobin. A rough guess may also be made as to the number of leucocytes, and the necessity for an actual count determined. The rapidity of coagulation may be arrived at by noticing how long it takes for threads of fibrin to

appear in the film, and the number of the blood-plates may be approximately reached. The presence of a number of small misshapen red corpuscles, or a diminution of hæmoglobin content, will indicate the necessity for both an actual count of the corpuscles and an estimation of their hæmoglobin. The standard of health is 5,000,000 red corpuscles, and 8000 to 10,000 white corpuscles in 1 cmm. of blood. Their enumeration is made with the help of a hæmocytometer. The Thoma-Zeiss, or a similar instrument, is the one most used for this purpose, and the whites may be counted at the same time as the reds, or separately, when two pipettes instead of one are used: one for the reds, in which the blood is diluted 100 or 200 times, and another of larger bore for the whites, in which the blood is diluted ten times. The student is urged to practice the enumeration in both ways until he has gained a considerable measure of dexterity. Full instructions are generally given with the instrument, and may also be obtained in all books on Clinical Methods. The estimation of the amount of hæmoglobin is carried out by means of a special instrument, known as a hæmoglobinometer. Several forms are in use—Gower's, Von Fleisch's, Oliver's and Haldane's. The two last are undoubtedly the best, and the most accurate. Oliver's is well known and well tried, but has the disadvantages of being expensive and of requiring artificial light, while Haldane's is a newer and cheaper instrument, which can be used in daylight, and is said to be equally accurate. Particulars of the method of use are supplied with the instruments. The standard amount of hæmoglobin in health is 100. If the amount of hæmoglobin present in any case be put as the numerator, and the percentage of the number of red corpuscles as the denominator, a fraction is obtained which expresses the hæmoglobin content of each corpuscle, and is conveniently spoken of as the colour index. Thus in health the amount of hæmoglobin is 100, and the percentage number of red corpuscles also

100, therefore $\frac{1.00}{1.00}$ or 1 is the colour index of each red cell. Other films of the blood must be fixed by heat, or by alcoholic formalin or other fixing agent. They are then stained with a combination of an acid and basic dye, such as eosin and methylene blue, Ehrlich's triacid stain, &c., and the characters of both the red and white cells further examined. The red cells are strongly acidophile on account of their contained hæmoglobin. Normal red cells, 7 to 8 μ in diameter, staining uniformly with acid dyes, e.g., red with eosin, are called *erythrocytes*; if larger than normal, 10 to 12 μ or more, *megaloeytes*; if smaller than normal, about 4 μ , *microcytes*; if irregular in shape, *poikilocytes*; and if nucleated *normoblasts* or *meqaloblasts*, according to whether they are of the size of normal reds or larger. The erythrocytes are the only form met with in health, the other forms appearing in certain abnormal states.

There is another abnormal condition which must be noticed, viz., where the red cell tends to take up both the acid and the basic stain, either diffusely or as blue dots on a red ground. This is described as *polychromatophilia*, and is looked upon as indicating degenerative changes within the red cells.

The white cells also give definite staining reactions, the nucleus taking up the basic dye, and the protoplasm the acid dye. Further, many of the cells possess within their protoplasm definite granules, which were formerly spoken of as *acidophile*, *basophile* or *neutrophile*, according as they take up the acid, the basic dye, or remain unstained. The term *neutrophile* is now largely dropped, as further observation has shown that these so-called neutrophile granules are really acidophile, although much more feebly so than the first described acidophile granules. The latter always stain strongly with acid dyes, such as eosin, and are therefore called *eosinophilous*, while the so-called neutrophile granules usually remain unstained when the film is stained with eosin and methylene blue, but stain

distinctly with the triacid stain. The leucocytes were formerly distinguished from one another by their nuclei, and spoken of as uninucleated or multinucleated, but it is now considered to be more accurate to distinguish them by their granules.

Two primary divisions thus appear, namely, hyaline leucocytes and granular leucocytes. The first division is called hyaline because the protoplasm shows no granules. It contains about 24 to 28 per cent. of all the leucocytes in normal blood. Two apparently different kinds of cells are recognised in this group—the lymphocyte and the large mononuclear cell, though some consider them to be modifications of the same cell. The former numbers 22 to 24 per cent., while the latter numbers only 2 to 4 per cent., of all the leucocytes. The lymphocyte is usually small, 5 to 8 μ , has a single round deeply-staining nucleus, with a thin rim of protoplasm around it, which also takes up the basic dye deeply or the acid stain faintly according to the combination of stains used. Larger forms are occasionally seen in disease, particularly in lymphatic leukæmia, and then the nucleus is apt to stain more feebly, the rim of protoplasm to be large, and to stain faintly. They thus approach closely to the large mononuclear leucocyte (10 to 15 μ or more), where both the round central nucleus and the plentiful protoplasm stain comparatively faintly. The second division of leucocytes (“the granular”) is further divided into two groups—the acidophile, or oxyphile, and the basophile. The acidophile is likewise subdivided into two, viz., the *finely* granular, and the *coarsely* granular. The finely granular leucocytes (10 to 12 μ or more) were formerly called neutrophiles. They number about 70 per cent. of all the leucocytes in normal blood. Their nucleus stains deeply with the basic dye, and the protoplasm feebly with the acid dye. The nucleus is twisted or divided in two or more pieces, generally,

however, united to one another by thin strands, hence the name polymorphonuclear, by which they are generally now known. The protoplasm contains the fine granules in large numbers. Most of the granules are very small, but they vary considerably in size, and some few are even as large as some of the granules met with in the coarsely granular leucocytes. This latter group—the coarsely granular ones—contains but a small percentage number (2 to 4) of the total leucocytes in the normal blood. Their nucleus may be single, lobed, or divided, and their protoplasm is rich in varying sized granules, mostly large, which stain deeply with eosin, and hence they are generally called *eosinophilous leucocytes*.

In certain diseases, never in health, the blood may show another form of leucocyte, known as a *myelocyte*. Its size is generally large (12 to 20 μ), although smaller forms also are always present. Its nucleus is single, round, rarely lobed, mostly eccentric, and stains feebly. Its protoplasm is studded with granules, mostly of small size, like those of the polymorphonuclear leucocytes, and like them with a feeble affinity for acid dyes. They stain very feebly or not at all with eosin, but well with the triacid or Jenner's stain. While this myelocyte is met with in quite a number of diseases it is especially characteristic of spleno-medullary leukaemia. A variety is occasionally seen in which the granules are coarse and stain deeply with eosin. It is called the *eosinophilous myelocyte*. The acidophile or oxyphile leucocytes thus include three forms—the polymorphonuclear, the eosinophilous, and the myelocyte. The other great division of granular leucocytes—the *basophile*—includes only one form, which is rarely seen in normal blood (never more than 0.5 per cent. of the total number of leucocytes), although it may be present in considerable numbers in disease, e.g., spleno-medullary leukaemia. Its nucleus is round, notched, lobed or looped, and stains feebly with basic dyes, while its plentiful protoplasm

contains a varying number of granules of irregular size, which show an affinity for the basic dyes, and thus stain blue with methylene blue. The average number of leucocytes in normal blood is about 8000 in 1 cmm. When it exceeds 10,000 to 12,000 we speak of the condition as a *leucocytosis*. This term really means an increase of all the leucocytes met with in the blood, and as the count is made from the fresh blood no differentiation between the different varieties of leucocytes is possible. To obtain this, it is necessary to fix and stain films. Leucocytosis occurs both physiologically and pathologically. The former is always moderate in amount, rarely exceeding 15,000. It occurs during digestion, after violent exercise, and during pregnancy, &c. The latter (pathological leucocytosis) is met with in disease. Strictly speaking, the term pathological leucocytosis means an increase in the total number of leucocytes, both normal and abnormal, irrespective of variety, occurring in disease; but it is usually restricted to signify an increase in the polymorphonuclear variety, and its presence or absence is often of great diagnostic importance. It occurs in a number of pathological conditions, among which the suppurative processes stand out prominently. It may enable the observer to be certain, when he would otherwise be doubtful, of obscure suppurations. It is thought that the toxins of the pyogenic cocci stimulate rapid production of polymorphonuclear leucocytes in the bone marrow, and thus a sudden overflow into the blood, and from the blood into the area of suppuration. If this be true, it becomes clear that it is not alone in suppurations, but in all inflammatory processes, that a leucocytosis tends to appear. If the poison be small in amount, the leucocytes will cluster into the affected part, but there will be little or no leucocytosis in the circulating blood, since the bone marrow is not greatly stimulated to over-production, and the escape of leucocytes from the blood keeps pace with their increased supply from the bone marrow.

All degrees of leucocytosis may thus occur in inflammatory processes. A high degree of it is usually seen in lobar pneumonia affecting vigorous adults, where the count may be 50,000 or more. It is not the poison alone which determines the leucocytosis. The system must react to the poison in order to produce it, and thus in a way it is a measure of the power of the body to react to a severe dose of the poison. A large dose of poison in a body capable of and giving a strong reaction results in a high degree of leucocytosis, probably nature's attempt to get rid of the poison. It may thus become of value in prognosis, for the outlook will be obviously graver in a severe infection with no leucocytosis than in one with a pronounced leucocytosis. It is not alone the toxins of the inflammatory and pyogenic bacteria which tend to be followed by a leucocytosis, for the toxins of many other organisms, as well as other substances, both organic and inorganic, also produce it; but not the toxins of all, for those of tuberculosis, measles, influenza, typhoid fever, &c., do not do so; indeed, there may be an actual diminution in the number of leucocytes, a *leukopenia* as it is called, in these and other diseases, of which some splenic anæmias are good examples. A pronounced leucocytosis also occurs after severe hæmorrhage. When the lymphocytes are increased in numbers, the term lymphocytosis is used. It may be absolute, *i.e.*, an increase in the lymphocytes alone, when the total count of leucocytes is above the normal; more usually it is relative only, when the lymphocytes are increased, although the total count of leucocytes is not raised. This occurs in infancy, where the lymphocytes number 50 to 60 per cent. of the normal leucocytes, steadily diminishing in number with age till they reach the 24 to 28 per cent. of adult life. It also occurs in many diseases, among which may be mentioned rickets, many of the infectious diseases of children, pernicious anæmia, &c.

An increase in the number of eosinophilous leucocytes is called *eosinophilia*. It occurs in most cases of spleno-medullary leukæmia and was formerly thought to be restricted to this disease, but it is now known to occur in a variety of other diseases, notably asthma, pemphigus and some other skin diseases, syphilis, gout, and intestinal parasites, *e.g.*, trichinosis, anchylostomiasis.

The above description of the corpuscular elements of the blood has been given in some detail, as it is essential for a proper understanding of the changes occurring in the so-called diseases of the blood. No attention has been given to the blood-plates, as they have not so far been shown to have any pathological or clinical significance. A convenient, though imperfect, method of dividing blood diseases is to place them into two great groups, according as they affect mainly the red or the white cells of the blood. We do not yet know sufficient about the changes in the blood-plasma to make use of them as a means of classification. The red or white cells may be affected either in the way of an increase or in the way of a decrease. An increase in the number of the red cells, *i.e.*, a plethora, is of comparatively rare occurrence and of slight or little known clinical significance, whereas a diminution in their numbers, *i.e.*, an anæmia, is of extremely common occurrence and much clinical significance. In the case of the white cells the opposite holds good, an increase in their number, *i.e.*, a léucocytosis, being much commoner and of better known clinical significance than a diminution, *i.e.*, a leukopenia. The anæmias are at the present time roughly divided into primary and secondary, according to whether an adequate cause is absent or present. The anæmias which are present in cases of tubercle, cancer, &c., are examples of secondary anæmia, because the primary tubercle, cancer, &c., adequately account for the subsequent anæmia, whereas the anæmia of chlorosis and pernicious anæmia are spoken of as primary, because there is no prior lesion in the body in these cases

which can be considered as an adequate cause of the anæmia, and we are compelled to refer its explanation to some fault in blood formation or blood destruction. Faults in blood formation may arise in a variety of ways. The most obvious is imperfect nutrition, either from imperfect food supply or from deficient assimilation, or there may be some abnormal drain upon the blood, either of all or of some of its constituents. Thus a hæmorrhage may take away a part of the blood and constitute an anæmia for such a time as it will take the blood-formers of the body to make up the loss. If the hæmorrhage be oft repeated, even though the loss of blood each time be small, as in hæmorrhoids, epistaxis, &c., the anæmia may become serious and lasting. Again, only some of the constituents of the blood may be lost, as in various intestinal fluxes, e.g., cholera, dysentery, &c., or in kidney disease with albuminuria. On the other hand, faults in blood destruction are also potent factors. There is constant destruction and renewal of blood going on physiologically; thus it is probable that the red blood-corpuscles have normally an average age of from only a fortnight to three weeks. When certain abnormal substances, notably the toxins of micro-organisms, or the chemical poisons generated by disordered digestion, or glandular secretions, find their way into the blood, the physiological limits of blood destruction may be surpassed and the processes of blood renewal interfered with. The morbid agent, whatever its nature, may cause chiefly a rapid destruction of red cells beyond the power of the blood-formers to remedy, even though these may be at the same time stimulated into unusual activity. The extent of the anæmia thus produced will depend upon the nature and virulence of the particular agent at work in each case, and doubtless also its action though mainly in one direction is not entirely so; and there may be in all cases an influence excited both on blood formation and blood destruction, though its most manifest effect is either upon the one or

the other. All forms of anæmia, whether primary or secondary, can be referred more or less fully to these two divisions, and as our knowledge increases we may be able to do this still more fully, and even to do away altogether with the use of the term primary anæmia. Anæmia is generally held to begin when the red blood-corpuscles do not exceed 4,000,000 per cmm., to become grave when they do not exceed 2,500,000, and fatal at 375,000 per cmm.

SUB-GROUP (a)—*THE PRIMARY ANÆMIAS.*

CHLOROSIS.

DEFINITION—A common primary anæmia of the female sex, most commonly seen in young girls, characterised by a low colour index, and, later, diminution in the number of the red cells, and by being very amenable to treatment.

Etiology.—A number of causes have been brought forward to explain its production, among which may be mentioned heredity, ovarian or uterine derangement, imperfect food, bad hygienic surroundings, physical fatigue, &c., but all of them break down when thoroughly examined into. The disease has certainly a tendency to run in families, and there is probably in all or most cases an innate tendency to the disease. Sexual derangement is common in many, and imperfect development of the sexual organs, as well as of the heart and blood-vessels, have been found in some cases; but the cases of chlorosis which show no such derangement or hypoplasia are far more numerous than those that do. Again, comparatively few individuals of the many exposed to defective food and hygiene, excessive fatigue, nervous shock, &c., become chlorotics. Its causal relation to constipation, through a consequent intestinal auto-intoxication, while it is supported by the frequent history of constipation in chlorotics, is, on the other hand,

contradicted by its as frequent absence. Further, the remarkable and rapid improvement which nearly all chlorotics show under iron and appropriate treatment tends to indicate that all these causes are really subsidiary to some underlying fault in the blood common to them all—in all probability some fault in blood formation.

Pathology.—I. CHANGES IN THE BLOOD.—(1) *The Red Cells.*—The most striking and characteristic change is a diminution in the colour index. Few, if any, of the other forms of anæmia ever show this so strikingly. It arises, of course, from a diminution in the amount of hæmoglobin out of proportion to the diminution in the number of the red cells. Fresh films show the red cells to be noticeably paler, but in the early stages of the great majority of cases they are neither much diminished in number, nor reduced in size, nor misshapen. In the later stages, the diminution in number generally becomes more marked, the number falling from about 4,000,000 to 2,500,000, which is a common average, or lower, even to 1,000,000, below which figure it rarely falls. Coincident with this fall, the hæmoglobin content becomes still further reduced, and the corpuscles become smaller and misshapen, microcytes and poikilocytes being common. A few normoblasts may also appear. (2) *The White Cells.*—They are not absolutely increased or altered, though they are relatively increased owing to the diminution in the number of the red cells.

2. CHANGES IN GENERAL NUTRITION.—These naturally follow upon the anæmic state of the blood. The skin and mucous membranes (lips, gums and conjunctivæ) are pale. The subcutaneous fat may increase. The muscles become flabby, particularly the heart, which may be dilated. The respiratory value of the blood being diminished, dyspnœa follows the least exertion. Œdema, chiefly of the feet, ankles and legs, is very frequent, owing partly to the enfeebled heart and partly to the anæmia. Thrombosis may occur in

any of the veins of the legs, but it is rare, or in the cerebral sinuses, where it is still more rare.

Symptoms.—These may be readily inferred from the changes in the blood and tissues of which we have just spoken. The pallor shows itself most distinctly in the conjunctivæ, in the lips, gums, tongue, lobes of the ears, and matrices of the nails. Sometimes the general pallor of the face is very marked, giving it a peculiar wax-like hue, with frequently a greenish tinge, hence the name chlorosis. Shortness of breath is an early symptom. It comes on especially after exertion, however slight, such as mounting steps, &c. There is disinclination to exertion, particularly in the morning, and the patient has generally slept heavily and awaked unrefreshed. She feels unready and unwilling for the day's work, but often improves as the day advances, because she draws upon her reserves of nervous strength and thus approaches nearer and nearer to the inevitable breakdown. The area of cardiac dulness may be increased, and there may be a systolic pulmonary or a systolic mitral murmur, or both, due to dilatation of the mitral orifice and feeble contraction of the cardiac muscles and the anæmic state of the blood. There may be palpitation, and there is often a loud murmur ("bruit de diable" or humming-top murmur) over the jugular vein at the root of the neck. The œdema of the legs appears first over the maleoli and dorsum of the foot, and is most marked at nights, generally disappearing by the morning. There is generally loss of appetite and some epigastric pain, which may be due to indigestion or to a gastric ulcer, which is not an infrequent complication. There may be vomiting. Constipation is very often present, and amenorrhœa is a very common symptom. Headache and giddiness are often complained of.

Diagnosis.—The appearance of the patient is often so distinctive that the diagnosis is almost ready made, but care

must be taken to exclude all other disease, for various forms of secondary anæmia, *e.g.*, tubercular, may otherwise lead to mistakes being made. The leading symptoms—viz., the pallor of the skin and mucous membranes, the breathlessness on exertion, the headache and giddiness, the amenorrhœa, and the absence of wasting or primary focal disease—make the diagnosis fairly easy, and examination of the blood sets any doubt at rest.

Prognosis.—This is generally very favourable, recovery taking place after a few weeks under appropriate treatment. Relapses are common, and some few cases prove very untractable.

Treatment.—The existence of a causal factor must be investigated, and if recognised its elimination should be attempted. Iron must be given, and by preference the ferrous carbonate, and the bowels should be kept regular by saline purgatives. The diet should be simple, nutritious and easily digested, and may be aided by the administration of cod-liver oil or cream. Country air and plenty of sunshine are of the greatest value. Care must be bestowed on the heart, and it is a good rule to investigate whether any dilatation has occurred, and if it has, to begin treatment by confining the patient to bed until such dilatation has disappeared.

PERNICIOUS ANÆMIA.

DEFINITION.—That form of much rarer primary severe anæmia which is most frequently seen in middle age, characterised by slight jaundice, by great diminution in the number of the red cells, but by a high colour index and by steady progression (usually) towards death.

Etiology.—The causation of this condition is still obscure. Steady and persistent loss of blood may cause

it, or at least may cause a condition closely resembling it in every respect except in the one fact of a recognisable source of previous blood loss. The *anchylostoma duodenale* or *bothriocephalus latus* may in this way produce a condition differing from ordinary pernicious anæmia only in showing a low colour index and a small amount of poikilocytosis; and other causes may act in the same way, for the blood loss may be due to an intestinal flux, to an atrophy of the stomach, to malignant disease, &c. These forms are spoken of as secondary pernicious anæmia, in order to distinguish them from the primary forms, in which no such mischief can be detected; and it is natural to scrutinise them carefully with the aim of discovering, if possible, something common to them all capable of explaining the blood condition. An actual loss of blood is not present in all, though it is in many. In some there are the conditions which will lead to defective blood formation rather than to its loss. But apart from such blood influences as these, it is obvious that another suggestion is possible, and even reasonable. The parasite may take away blood it is true, but it may also produce a toxic substance whose absorption into, and subsequent action upon, the blood is of far more importance. This toxic agent may be the real cause, the actual blood drain in whatever form it exists merely lessening the natural resisting powers of the body and so hastening the process. The presence of a toxic agent not only cannot be excluded but is even highly probable in all the forms of secondary pernicious anæmia. And if so, why not in the primary forms? Experience shows us that in undoubted infective disease the source of infection is often hidden, and primary pernicious anæmia may readily be another instance. This hypothesis is supported both by analogy and by many of the characteristics of the disease. It has been ably advocated by Dr Wm. Hunter of London and many continental observers. The poison may be directly or indirectly of

bacterial origin or produced by faulty gastric or intestinal digestion; but however produced, its absorption by the blood leads to an excessive destruction of the red blood-corpuscles. Hunter believes that the liver is the main seat of this destruction. Pernicious anæmia is, according to this view, essentially due to excessive hæmolysis, as distinguished from chlorosis, which is more probably due to defective hæmogenesis. This view is, while widely held, not universally accepted. There are many who still believe in the older view that defective hæmogenesis is the starting point and essential cause of this disease also, the excessive hæmolysis, which is a constant feature, being regarded as a result. Stockman holds a third view, viz., that the disease is really due to repeated small capillary hæmorrhages, which, being internal, are hidden, and hence that there is no such thing as a really pernicious anæmia. He thinks that the large amount of iron in the liver so characteristic of pernicious anæmia is due to such hæmorrhages occurring in the intestine.

Pathology.—I. CHANGES IN THE BLOOD.—(1) *The Red Cells*.—The most striking change is a marked diminution in the number of the red cells, with, at the same time, a retention, or rather an increase, of their hæmoglobin content. The colour index is consequently almost always *above normal*. The average number of red cells in well-established cases of the disease is about 1,000,000. Death generally follows when the number falls below 400,000, and the usual count at death is between 600,000 and 300,000. In addition to this diminution in number there is also a marked irregularity in size and shape of the red cells, many being oversized, others undersized, and many of irregular shape—megalocytes, microcytes and poikilocytes respectively. The megalocytes, while often much more numerous, rarely form less than 33 per cent. of all the red cells present, and this probably

explains the increased hæmoglobin content; they may be very scanty during remissions. The microcytes are often very scanty and are less characteristic. The poikilocytes, on the other hand, are commonly present in considerable numbers, the pear-shaped being the most prevalent. Nucleated red cells are always present (most probably), both normoblasts and megaloblasts, though they may be very few in number, requiring prolonged searching of many microscopic fields to find them. In other cases they are fairly numerous, and in all cases in which they are sufficiently numerous to make the comparison it will be found that the megaloblasts outnumber the normoblasts, and, indeed, in typical cases the normoblasts are either excessively few or altogether absent, whereas the megaloblasts are fairly common. They are usually more numerous in severe cases. Polychromatophilic changes are also very frequently seen in many of the red cells, both nucleated and non-nucleated, being nearly constantly present in the megaloblasts. Another constant and characteristic feature is the diminished tendency to rouleaux formation in fresh drops of the blood, due partly to their diminished numbers and partly to certain physical and chemical changes within themselves. (2) *The White Cells*.—There is generally a distinct diminution in the number of leucocytes—a leucopenia, which progresses with the disease. A count of 4000 to 5000 is a common average, but it may be much less in extreme cases. The diminution is chiefly in the neutrophils, so that there appears to be a relative increase in the lymphocytes—a relative lymphocytosis, slight in mild cases, more marked in severe cases. Abnormal leucocytes may also appear, viz., myelocytes in small numbers.

2. CHANGES IN GENERAL NUTRITION.—The skin and mucous membranes are pale and slightly jaundiced. The subcutaneous fat is not diminished. There is no noticeable loss of flesh, but the muscles are flabby and the heart

usually dilated. Small hæmorrhages in the skin and mucous membranes are frequent. Œdema is common about the feet and ankles. Fatty degeneration gradually appears and is generally prominent in all the organs, especially the heart, at death. The liver, though often normal in size, may vary. It is usually fatty, but its most striking feature is the presence of a large amount of iron in the outer two-thirds of its lobules. This iron pigment is also present in excess in the spleen and other organs, particularly the kidney, but in none of them to anything like the same extent as in the liver. Hunter's analyses place the amount of iron found in the liver in this disease to be on the average seven times that found in any other disease attended with anæmia. The bone marrow is usually red and soft, like lymphoid tissue, and shows great numbers of nucleated red cells, particularly megaloblasts. A sclerosis of the posterior or postero-lateral columns of the cord has also been found in some cases.

Symptoms.—The anæmic and slightly jaundiced hue of the patient presents a characteristic picture. The onset of the disease is generally very gradual and insidious, and it is accompanied by feelings of lassitude, disinclination to work, both physical and mental, a breathlessness and exhaustion on any exertion. Digestive troubles, vomiting, diarrhœa, &c., are usually present, and may become very marked. Palpitation and irregularity of heart action are common. Cardiac bruits are common. The temperature often rises in an irregular way to 102°F. or even more. There is a great tendency to small hæmorrhages in the skin, mucous membranes and particularly in the retina, where they can generally be seen with the ophthalmoscope. The urine is usually of a low specific gravity, and may be pale or highly coloured. In the latter cases it has been shown to contain pathological urobilin, which is pointed to as supporting the hæmolytic theory of the disease. It is also said that this

character of the urine coincides with paroxysms of the disease, which often runs a remittent course. The mind is apt to wander and become torpid. There may be tinglings, headache, &c. Great prostration gradually supervenes, and death follows from asthenia in the absence of some intercurrent disease.

Diagnosis.—The appearance of the patient and the examination of the blood make the diagnosis easy in most cases. While all the characters of the blood should be considered, most reliance should be placed upon the high colour index, the presence of many megalocytes, and the relatively large number of megaloblasts present.

Prognosis.—It is generally a steadily-progressive disease ending in death. The average duration may be taken to be from about one to two years, but some cases improve or remain stationary, or nearly so, for long intervals—months to two or three years. These remissions have become more frequent since the treatment of the disease by arsenic, but Byrom Bramwell's case, which remained fairly well for twelve years, is still exceptional.

Treatment.—It is obvious from a reference to the etiology of this disease that certain fairly definite causal conditions may be present, and these should be searched for and carefully treated. Whether we favour Hunter's view or not, oral sepsis should, if existent, be attended to, and, further, intestinal antiseptics such as salol or β -naphthol are worth a trial. Arsenic is, and for long has been, the most satisfactory remedy, and may be administered in 3 to 15 minim doses thrice daily *with food*. Iron is generally of little use, but should be given until one is convinced that it is not benefiting the patient. Rest in bed with plenty of fresh air and sunlight, nutritious food, and exercise in the form of massage may be considered the best general

rules to follow. Transfusion with defibrinated human blood (3 to 6 oz.) mixed with phosphate of soda solution has proved of value, and oxygen inhalations are beneficial in severe cases. Red bone marrow is very disappointing in its results, and causes indigestion.

SUB-GROUP (β)—THE SECONDARY ANÆMIAS.

This includes all forms of anæmia arising in the course of another disease, *e.g.*, tubercle, cancer, renal disease, after-hæmorrhages, and so on. The severity of the anæmia will, in these cases, depend upon the severity of the primary disease, and hence a periodic examination of the red cells is often an important guide in the progress of the case.

(1) *The Red Cells.*—The number of the red cells will therefore vary considerably from 4,000,000 in mild to under 1,000,000 in severe cases. The total amount of hæmoglobin will be correspondingly diminished; but their hæmoglobin content is, as a rule, unaltered, the colour index being usually normal. Both microcytes and poikilocytes may be comparatively numerous, but megalocytes are scanty. Nucleated red cells are fairly common in most of the forms. They are mostly normoblasts, and when megaloblasts also occur, as they do in some forms, *e.g.*, malignant disease, they are always less numerous than the normoblasts. Polychromatophilic changes may also be observed. (2) *The White Cells.*—They may be increased, diminished or unchanged, according to the nature, rapidity and severity of the poison and the reaction of the organism to it. The increase may be in the neutrophiles, as in post hæmorrhagic anæmias, &c., where the leucocytosis is rapid and often considerable (25,000 or more), or in the lymphocytes, as in rickets, measles, &c., where, however, the lymphocytosis is always slight, for in

none of them does it reach a degree properly termed pathological. In one disease alone does it ever do so, and that is lymphatic leukæmia.

Treatment.—After a severe hæmorrhage the number of red cells is rapidly restored to its normal figure, but in all cases of secondary anæmia the cause must be sought for and combated. Where a severe and persistent drain, especially upon the blood-plasma, is present, as in Bright's disease or protracted suppurations, the recognition and treatment of the anæmia present are most important. Poisons, whether inorganic (such as lead) or organic (such as the toxins present in many diseases), must be taken into account and antagonised. Iron is the great remedy for all kinds of secondary anæmia, together with plenty of fresh air, good food and rest from mental and physical work.

II.—*DISEASES AFFECTING THE BLOOD-FORMING GLANDS.*

SUB-GROUP (a)—*DISEASES AFFECTING MAINLY THE WHITE CELLS.*

This is not really an accurate grouping. No single method of arrangement is free from fault, but it is convenient to include within it at least three important diseases, viz., Leukæmia, Splenic Anæmia and Hodgkin's Disease. The first alone of these, viz., leukæmia, can justly be described as a disease in which the changes in the white cells form the main characteristics of the disease. In Hodgkin's disease, at least in its typical forms, there is no great departure from the normal in the white cells, but in some of its unusual forms there is, and there are

certain advantages in describing this disease here rather than under the lymphatic glands, where it may, indeed, more properly belong. In splenic anæmia there is mostly a marked leucocytosis, and further research may yet show that it and lymphæmia are more closely allied to one another than seems at present to be the case.

LEUKÆMIA.

Syn. Leucocythæmia.

DEFINITION—A disease characterised by a great increase of leucocytes in the circulating blood, associated with changes in the spleen, bone marrow or lymphatic glands.

Etiology.—The cause is still hidden. The disease may appear at any age, but is most frequent in middle life. Males are more frequently affected than females, and in some cases there is a marked hereditary relationship. Trauma, syphilis, rickets, or malaria have all been shown to exert a causative influence in certain cases. There are many suggestions in favour of an infective agent, and several micro-organisms (both bacterial and protozoal) have been put forward as the responsible agents. The evidence so far available in support of any of them is too slight to warrant further notice here. That it is, however, some toxic agent or agents which act upon the blood-forming organs seems highly probable.

Types.—Two types—the myelogenous and the lymphatic—are distinguished, according to the changes presented by the blood; but while the classical examples of each are easily classified in this way, there are others which partake somewhat of the characters of both, so that mixed types occur, or a case may in its earlier stages seem to belong to the first, and in its later stages the second.

Pathology.—1. BLOOD CHANGES—A. The Myelogenous or Spleno-medullary Leukæmia or Myelæmia or Myelocythæmia. The blood is usually lighter in colour, and coagulates slowly. (1) *The Red Cells*.—There is at first little or no diminution in the number of the red cells, but later, after the disease is well advanced, they usually number from 2,000,000 to 3,000,000, and may fall much lower in the last stages of the disease. The hæmoglobin is diminished much more rapidly than the corpuscles, so that the colour index falls as in chlorosis. In the later stages, however, this ratio may not be preserved, and the colour index may rise. Normoblasts generally appear in considerable numbers at an early stage, more so than in any other disease, and they mostly remain numerous throughout, though later megaloblasts in scanty numbers usually also appear, and then megalocytes, microcytes and poikilocytes are also evident. In some severe cases the picture presented by the red cells strongly resembles that of pernicious anæmia. (2) *The White Cells*.—It is here that the most characteristic changes are seen. There is a great, generally enormous, increase in the number of leucocytes—from 100,000 in mild, to 300,000 to 400,000 in moderately severe, and 600,000 to 1,000,000 or more in very severe cases. While 100,000 is here mentioned as a low count, it must not be inferred that it may not be still lower; indeed, our knowledge does not allow us at the present time to state any limit in this direction. Many inflammatory leucocytoses may give a higher count at times than a true leukæmia. The leucocyte count in cases of true leukæmia varies very greatly, not only in different cases but at different times in the same case. It may even fall to and remain at normal for considerable intervals of time. While all the different leucocytes normally met with in the blood are absolutely increased in numbers in the usual leukæmic count, stained films reveal that at least one abnormal form has appeared in such numbers as to disturb the relative proportions of all

the other forms. This is the myelocyte. The neutrophile myelocyte is the prevailing form, but the eosinophilous variety is also present in varying numbers in different cases. In chronic examples of the disease myelocytes are present in such numbers as to form a striking feature in the blood films. In 28 cases Cabot found that between 20 and 60 per cent. (average 35 per cent.) of all leucocytes present were myelocytes. In acute examples, on the other hand, which are very uncommon, they may be scanty, not exceeding in numbers and proportions those seen by Engel in fatal diphtheria, and by other observers in most acute infections. They are apt to undergo degenerative changes, involving pallor and vacuolation of their nuclei and loss of their granules, which make their recognition very difficult. Myelocytes, unlike the polymorphonuclear leucocytes, are said never to exhibit amœboid movement.

The method of escape of these two classes of cell from the bone marrow into the circulation must therefore be essentially different. The escape of the polymorphonuclear leucocytes is an active one. By virtue of their amœboid movement they leave the marrow and wander into the circulation, whereas the myelocytes are passive and must be swept by fluid waves into the blood. Their numbers usually dwarf the polymorphonuclear forms, and make them to appear less numerous than usual in the stained films, so that, as a rule, the more numerous the myelocytes, the less numerous the polymorphonuclear leucocytes. Cabot puts the proportion of the latter at from 17 to 72 per cent. (average 46 per cent.). It must be remembered, however, that their total numbers, like those of the other leucocytes, are always greatly increased. Degenerative changes in these cells are also common. Their nuclei may become very pale or completely fragmented and very dark or vacuolated, and their protoplasm may lose its granules. The eosinophilous leucocytes are also increased in total numbers, so as in most cases to

greatly exceed those found in any other disease (3000 to 100,000 per cmm.—Ehrlich), but their normal proportions (2 to 4 per cent.) to other forms of leucocytes are not increased except in a few cases. In the acute examples they are very scarce. A large proportion of the eosinophilous cells in the majority of ordinary cases (chronic) are eosinophilous myelocytes. Eosinophilous cells are indeed always more or less typical of chronicity. The lymphocytes are usually markedly diminished in proportion (average 10.6 per cent.—Cabot), but vary greatly in different cases, and at different times in the same case. Large mononuclear leucocytes are seen in most cases, but their numbers vary considerably. Generally the more purely myelogenous the disease, the scantier they are. Mast cells are usually so markedly increased as to constitute a reliable diagnostic feature. The more chronic the case the more plentiful they are, as a rule, and they may even outnumber the eosinophilous cells.

B. Lymphatic Leukæmia or Lymphæmia. (1) *The Red Cells.*—They present much the same characters as in myelæmia. Nucleated red cells, however, are usually very scanty and may be absent. (2) *The White Cells.*—The blood count is often below, but sometimes it is much above, the ordinary average of myelæmia, some of the highest counts of all having been got in this variety. This increase is due to a great abundance of lymphocytes. It is the ordinary small uninucleated cell (7 to 8 μ) which is affected, and it usually forms 80 to 90 per cent. of all the leucocytes present. In a small proportion of cases the lymphocytes are mostly much larger (15 μ or more), particularly in cases running a rapid course and in children. These large mononuclear cells vary considerably in the depth of stain taken up by the nuclei, while their protoplasm almost always stains very feebly. Beyond indicating in all probability an acute stage of the disease, it is impossible at present to say if they have any other significance.

The form which looks like a large lymphocyte (deeply stained nucleus and deeply stained small amount of protoplasm) has never been seen in adults except in very acute cases, whereas the other form (the large mononuclear cells, with paler nucleus and more abundant paler protoplasm) has been seen both in acute and chronic forms, though more often in the former. Further, cases have been described as acute which show only the small lymphocyte. Myelocytes, eosinophilous cells and mast cells are usually absent or extremely scarce. Polymorphonuclear leucocytes are also comparatively scarce. It is to be remembered that lymphæmia is much rarer than myelæmia, and that most, though not all, of the acute cases belong to it. An important practical point is the distinction between cases of chronic lymphocytosis, other than lymphæmic, and cases of true lymphæmia; and while this is generally easy, it is not always so, for it is not at present possible to say what is the lowest proportion of lymphocytes which constitutes a true lymphæmia. All the other features must be taken into account, and even then the differentiation may remain, at least for a time, impossible.

2. THE VISCERAL CHANGES.—(1) *The Bone Marrow*.—This is distinctly affected in all cases of myelæmia and in many cases of lymphæmia. In the latter its distribution is sometimes very irregular, much more so than in the former, and it may thus escape detection. Several cases are, however, reported in which it was apparently unaffected. In all cases the nature of the change is the same, viz., a cellular hyperplasia, and the variety of cell affected is the same as in the blood: thus in myelæmia it is the neutrophile myelocyte and in lymphæmia it is the lymphocyte. Many of these cells are seen in mitotic division or in various stages of degeneration. This rapid multiplication of the myelocyte or lymphocyte, or both, in mixed cases is the essential change in the disease. It is seen particularly in the marrow of the ribs, sternum, vertebræ, &c., and

along with it there is an extension of lymphoid marrow throughout the shafts of long bones. To the naked eye the marrow may in acute cases appear almost puriform, though less diffuent than pus. In chronic cases it may in the early stages show a fleshy tint, but later it is always light-coloured and firm.

(2) *The Spleen*.—This organ is enlarged in most cases, but it is in the myelogenous variety in which the enlargement is most striking. The increased size may be enormous, and the organ may weigh as much as 18 lb. The enlargement is uniform: its capsule is smooth except for simple inflammatory adhesions, which are more common in the later stages. The organ is firm, and in enlarging preserves its normal shape. Hence it is easily palpated and recognised during life, the notch in its rounded anterior border being easily distinguished. On section it shows a pale fleshy or reddish-brown colour, and the Malpighian bodies are invisible. Occasionally infarcts of varying size are seen. Microscopically, the nature of the enlargement is seen to consist here, also, of a great cellular hyperplasia, involving large and small mononuclear cells. They choke the pulp sinuses and obscure the margins of the Malpighian bodies. They probably originate through rapid multiplication of the lymphocytes in these Malpighian bodies. Neutrophile myelocytes are often also seen in moderate numbers in myelæmia, but not in lymphæmia, and are probably brought hither by the blood-stream. The pulp sinuses also contain an excess of ordinary red blood-cells and a few nucleated ones. This great lymphocyte cellular hyperplasia gradually gives rise in the later stages to much diffuse fibroid change. These appearances of the spleen, and particularly the absence of any sign of myelocyte formation within the spleen, have led to the suggestion that the enlargement of this organ is an entirely secondary feature, in more purely myelogenous forms at any rate, and caused probably by the mechanical sifting of red and white

cells from the circulation and subsequent inflammatory changes. In the lymphatic variety, on the other hand, the proliferation of lymphocytes is usually very marked, and the Malpighian bodies may, in many cases at least, be important primary sites of the disease.

(3) *The Lymphatic Glands*.—They are generally enlarged, particularly in the lymphatic variety, either along with the spleen or alone. They may grow to some size (that of a hen's egg), but usually remain soft and movable. The cervical, axillary, mesenteric and inguinal are usually most affected, but the lymph follicles of the tonsil, pharynx and intestine also partake in the process. The nature of the enlargement is the same as in the spleen—a diffuse hyperplasia of the lymphocytes, which show plentiful mitotic division. This enormous production of lymphocytes crowds the follicles and chokes the sinuses and supplies new leucocytes to the circulating lymph and blood.

(4) *The Liver*.—This organ is almost always enlarged. In the myelogenous variety, distinct whitish nodules of varying size or a diffuse whitish infiltration is usually seen by the unaided eye. They consist at first of masses of leucocytes, probably a white cell thrombus in a capillary, later they form a reticulum and take on the structure and arrangement of normal lymphoid tissue. In the lymphatic variety they seldom form definite lymphoid nodules, but are usually limited to a small round-celled invasion of the portal tracts and lobular capillaries. Normoblasts are also frequently found in the hepatic capillaries in myelæmia.

(5) *The Kidneys*.—They are often enlarged and pale. Great numbers of leucocytes crowd the capillaries, and may form definite large masses, as in the liver.

(6) *Other Organs*.—The capillaries of all the other organs of the body are more or less crowded with leucocytes, but definite lymphoid masses are rarely found in any of them.

Pathogenesis.—The exact nature of leukæmia cannot yet be formulated without leaving room for doubt, but the facts, so far as they are ascertained, seem to support the conclusion that it is a primary disease of the blood-forming organs, principally or exclusively affecting the bone marrow or the lymphoid tissues. In myelæmia the bone marrow is probably exclusively at fault, primarily at any rate, and the fault lies in the hyperplasia of the myelocytes. In lymphæmia not only the marrow, but any or all of the lymphoid structures of the body are at fault, the hyperplasia here affecting the lymphocytes and not the myelocytes. In some cases the marrow may be initially inactive and remain so during the course of the disease, some or all of the other lymphoid structures of the body showing the activity. The exact seats of activity probably differ considerably in number and degree in different examples of this disease. The new leucocytes are supplied to the circulation from these sources, viz., the bone marrow and lymphoid structures, and any multiplication within the circulation itself is comparatively insignificant. After they leave the circulation and become deposited in the viscera, as in the liver, they again multiply and proceed to form a tissue reminiscent of the normal lymphoid tissue of the body. The resemblance of this process to that of a sarcoma is striking, and has led to the suggestion that lymphæmia at any rate is merely a variety of lymphosarcoma. The characters of growth and metastasis shown by true lymphosarcoma differ too much from those of true lymphæmia to allow of this suggestion being accepted, and further light must be awaited before we can proceed further.

Symptoms.—In the ordinary classical case, the onset of the disease is very insidious. Failing health and strength, want of appetite, irregularity of the bowels, particularly attacks of diarrhœa, are among the first symptoms: or the patient may complain of heaviness or swelling in the

abdomen, though this usually escapes notice for a considerable time; indeed, the spleen may be found to be of great size the first time the patient consults the physician, reaching as low as, or even lower than, the umbilicus. Its firmness, the smooth surface, the "notch" on the rounded anterior border, the continuity of the tumor and of the area of dulness into the splenic region enables the observer to recognise with ease that the tumor is the spleen. There is no pain in the region of the abdomen, as a rule, in the earlier stages of the disease, which accounts, perhaps, for the patient failing to notice the gradual enlargement of the abdomen, or thinking it unimportant if it has been noticed. There may be swelling of the cervical glands, with or without abdominal enlargement. Bleeding from the nose is also common in the initial stages, and may cause much trouble, owing to the feeble coagulating power of the blood. Hæmorrhages in other regions, except perhaps the uterus, do not come on till later, when, however, they may be very frequent and very numerous, affecting mucous, serous and every surface. Scattered hæmorrhages in the retina are very common (leukæmic retinitis), which interfere with vision when they happen to affect sensitive areas. Hearing may be affected in the same way. A cerebral hæmorrhage may cause apoplexy and death. There is no special change in the urine so far as is yet known, but there is excess of urea and uric acid. There may be priapism, supposed to be due to clotting in the corpora cavernosa. Slight feverish attacks (102 to 103°F.) are also common. The gastro-intestinal symptoms usually remain prominent throughout, and though the progress of the disease may appear to be arrested at intervals, it is usually steadily downwards. Anæmia, which is rarely present at first—indeed, the patient may look remarkably well—appears in time and steadily progresses, bringing in its train lassitude, failing strength, headache, fainting, and a failing circulation. Emaciation becomes noticeable and then marked, and if hæmorrhages be

numerous the case may come to show all the signs of pernicious anæmia. If not cut off earlier by some intercurrent disease, the increasing asthenia becomes fatal in time. The usual duration of such a case from the time it first comes under observation (probably a long time after its commencement) is from two to three years; but other cases may be much more rapid, and some few acute cases (almost always lymphæmic) may prove fatal in seven to thirty or sixty days.

Diagnosis.—This is easy in typical cases, but may be difficult or impossible in aberrant forms. In all cases, examinations of the blood, oft repeated, are chiefly to be relied upon, and, owing to increased knowledge and better technique, their results are likely to afford more complete information in the future than was so often possible in the past. A careful postmortem examination of the bone marrow and lymph-glands may clear up certain cases which defy a sure diagnosis during life. The two diseases most liable to lead the observer astray are splenic anæmia and Hodgkin's disease, and the examination of the blood mostly sets him right at once.

Prognosis.—Most cases steadily progress and die after two to three years. Intermissions, however, are common, and the patient may remain fairly well, with no apparent progress downwards, for long and recurring intervals, even two years or more. It is very doubtful if any genuine case ever recovers, though some authorities hold that they do.

Treatment.—There is one remedy—arsenic—and if it fails we have little to fall back upon. Arsenic should be given at first in small doses *with food* and pushed until 10 minims, or even more, of the liquor arsenicalis are taken thrice daily. Under this treatment the myelocytes may

greatly diminish in number, and the spleen actually decreases in size. In acute cases, many of which are lymphatic in type, its success is more doubtful. Feed carefully and apply mercurial ointments locally over the spleen. Quinine has sometimes been found to be advantageous.

SPLENIC ANÆMIA.

This name as at present used probably covers more than one disease. It was used by Von Jaksch to designate a form of infantile anæmia presenting close resemblances to leukæmia, but distinguishable therefrom, and consequently sometimes called a form of pseudo-leukæmia. It has chiefly been met with in children between one-half and four years of age, who have had either rickets or syphilis, and hence its claim to be regarded as a primary blood disease is at least doubtful. The chief blood-changes are—(1) A grave anæmia, varying usually between 3,000,000 and 1,000,000 red cells and showing a great number of nucleated red cells; (2) a high and persistent leucocytosis (20,000 to 50,000, sometimes higher), in which the mononuclear cells have in most cases been slightly in the majority. Myelocytes in small numbers have been seen in some cases. The chief visceral changes are—(1) A marked enlargement of the spleen; (2) slight enlargement of the liver; and (3) occasional slight enlargement of the lymphatic glands. In some cases the anæmia is so profound that it simulates a pernicious anæmia, though the blood-changes generally make the distinction easy. There are never any leukæmic deposits in the viscera, and usually the case runs a very chronic clinical course, far more so than leukæmia, and recovery is frequent.

The term splenic anæmia has also been applied to cases of splenic enlargement with anæmia occurring in adults. They clinically simulate spleno-medullary leukæmia, from which they are easily separated by the simple fact that they show no leucocytosis, but generally, indeed, a leucopenia in all stages, except occasionally the later stages or after severe hæmorrhages, when a certain amount of leucocytosis may appear. The anæmia is not at first profound, but usually steadily progresses and becomes so in its later stages. The enlargement of the spleen is also steadily progressive, and, unlike leukæmia, is held to bear a primary and essential relationship to the causation of the disease, inasmuch as if the spleen be not excised the case is certain to steadily progress to a fatal issue. Cases have been recorded in which excision of the spleen has been followed by recovery, a procedure which has never, on the other hand, done the least good in leukæmia. While the clinical course is usually chronic and spread over three, four, or more years, some cases are much more rapid, and end fatally in less than one to two years.

Banti's disease is the name given to a form of splenic anæmia in which there is also cirrhosis of the liver, often of the hypertrophic variety. Its origin is obscure. It is characterised by splenomegaly, cirrhosis of the liver and anæmia. Some authorities doubt its claim to a separate entity.

Treatment.—Probably in most cases with leukopenia, and certainly in Banti's disease, splenectomy is the one hopeful treatment. Arsenic may be tried, but is usually very unsuccessful.

CHLOROMA.

DEFINITION—A little known but fatal disease allied to lymphatic leukæmia and lymphosarcoma, and characterised by the presence of greenish tumors in different parts of the body, and often by a lymphocytosis.

Etiology.—Only some twenty-seven cases have hitherto been described up to end of 1901. They have occurred mostly in male children. Tubercle was present in several cases.

Pathology.—Tumors of varying size and greenish-yellow colour have been found in different situations, such as the bones of the skull, particularly in the orbits and nose, vertebral canal (where they may compress the cord, causing ascending degeneration), anterior surface of bodies of vertebræ, sternum and skin. Metastases (green in colour) may be present in liver, heart, and kidneys. Microscopically, they consist of lymphoid tissue showing large lymphocytes, a reticular stroma, and often also roundish clusters of granules of a fatty nature, staining faintly with osmic acid and sudan III., lying outside the cells. The changes in the blood and blood-forming glands are important.

1. THE BLOOD.—(1) The red cells are diminished in number, sometimes as low as 1,000,000 per cmm. (2) The hæmoglobin is also greatly diminished. (3) Nucleated reds in small numbers are often present. (4) The leucocytes may or may not be absolutely increased (a count of 300,000 per cmm. has been got); but the medium and large lymphocytes are always increased, while the polymorphonuclears and small lymphocytes are always greatly diminished. Myelocytes may be present.

2. THE BLOOD-GLANDS.—(1) The lymphatic glands are usually all enlarged and green in colour. Histologically, they show a hyperplasia, and their sinuses contain large

lymphocytes similar to those of the tumors and the blood. (2) The spleen is enlarged and shows similar characters. (3) The bone marrow may show the green colour, and also contains large numbers of these large lymphocytes. The cause of the green colour is held by some to be due to the fatty granules, by others to a derivative of the blood pigment, and by others to a chemical substance allied to lipochrome. The heart is fatty. From these changes can anything be deduced as to the nature of the disease? If the tumors be primary and the blood condition secondary—the more probable view—the disease ought to be classed among the Lymphosarcomata; several German observers (Risel, Rosenbluth) incline to this view, and suggest the name chlorolymphosarcoma for the disease; whereas, if the reverse be the case, it is a blood disease, related closely to leukæmia, and other observers (Byrom Bramwell, Dunlop) support this view. The absence of an absolute lymphocytosis in some cases, and its appearance in others only a few days before death, support the first view.

Symptoms.—In addition to the changes in the blood, which can of course be detected during life, the skin and mucous membranes may show petechiæ, and the former also small greenish tumors. The cervical glands are often noticeably enlarged. There is a marked protrusion of one or both eyeballs, accompanied by pain. Optic neuritis and blindness come on later. Deafness also has occurred in most of the cases.

Prognosis.—It is rapidly fatal, usually within four months.

Treatment.—Nothing can be hoped for from treatment beyond the usual careful dieting and alleviation of symptoms. Arsenic may be tried but without much anticipation of benefit.

HODGKIN'S DISEASE.

Syn. *Lymphadenoma*, *Adenic*, *Adenia simplex*, *Pseudoleukæmia*.

DEFINITION.—A painless progressive enlargement, usually chronic, of the lymphatic glands and usually spleen, along with anæmia and fever.

Etiology.—There is very little known about the causation of this disease. In typical cases the disease is of untraceable origin and gradual development. It is commonest in the young, particularly males. In a number of cases there has been some antecedent inflammatory condition in the mouth, throat, nose or skin, which may have acted as a source of infection, and hence the suggestion has been made that the disease is due in some cases, if not in all, to some bacterial agent. Nothing, however, is definitely known about this, and we have to be careful lest we be misled by the analogy of certain infectious conditions which may produce a disease so closely resembling lymphadenoma in its clinical features as to make it almost, if not quite, impossible to distinguish the one from the other during life. Thus many cases of tubercular disease affecting the lymphatic glands may so closely simulate lymphadenoma as to be indistinguishable from it, except by histological or experimental proof of the presence of the tubercle bacillus. Other organisms, particularly the pyogenic forms, may act much in the same way, and it is probable that many of the cases described by writers as instances of acute lymphadenoma may really have been cases of acute infection of this nature. Some such cases, whose true nature has been recognised by their morphological changes and bacterial tests, have been put on record by observers. These are not the only difficulties which have to be met in saying what is and

what is not lymphadenoma, for though typical examples of the disease are easily distinguishable from lymphosarcoma on the one hand, and lymphatic leukæmia on the other, yet there are cases which seem to run by insensible gradations into both these diseases. All these considerations make the problem of the causation of lymphadenoma at the present time an extremely difficult one.

Pathology.—1. CHANGES IN THE LYMPHATIC GLANDS.

—A steady and progressive enlargement of some or most of the lymphatic glands of the body is the most striking character of the disease. It first affects the cervical glands of one side in the great majority of cases, though occasionally it may begin in another group, such as the groin. All or most of the members of the chain of glands are usually, though unequally, involved. The size of the enlarged glands varies, though it is rarely great in any individual instance, the size of a hen's egg being rarely reached or exceeded. In typical cases the enlarged glands are firm, and freely movable from one another and under the skin. In the exceptional cases of acute course the glands are softish, and may even give a sense of fluctuation. In the later stages of the disease it is not uncommon for the glands to become more or less adherent to one another and to the superjacent skin. The explanation of this adhesion is found in the enlargement of some of the glands first involving their capsules and then invading neighbouring glands and the skin. The nature of the enlargement is essentially a hyperplasia of the lymphoid cells. These are enormously increased, and later lead to the formation of an irregular fibrous stroma, so that those glands in which the disease is of long standing are often very fibrous, but they do not tend to suppurate or caseate. From the cervical glands of one side of the neck the disease generally passes, it may be after an interval of months or years, to those of the other side, and affects

them much in the same way. The glands of the axilla are next similarly affected, then follow those of the groin, abdomen, mediastinal, bronchial and pelvic groups. The other internal glands and masses of lymphoid tissue suffer in a similar way, but may not be detectable during life, either by inspection, palpation or percussion. This generalisation of the change in the lymphatic glands is characteristic of the disease. It may take weeks, months or years to accomplish it, and there may be almost no appreciable interval, or a very long one, in its passage from one chain of glands to another, and there may be great variety in the degree of affection which the different chains of glands, and the individual glands in each chain, may show in different cases.

2. CHANGES IN THE SPLEEN.—This organ is enlarged in the great majority of cases, but the enlargement is not great, the organ being rarely palpable more than two or three inches below the ribs, thus contrasting strongly with the very much greater enlargement which prevails in leukaemia. The enlargement is uniform and the organ preserves its shape, so that the characteristic anterior notch is generally easily felt during life. Further, in a great many of the cases the enlarged spleen shows a number of whitish-yellow masses, varying in size from a pin-head to a pea or bean or larger, often grouped together in clusters, scattered throughout the interior of the organ. They are not unlike small lymphatic glands, and have been likened to pieces of suet infiltrating the organ. They are masses of lymphoid tissue, consisting in their early stages mainly or entirely of lymphoid cells, but later, like the lymphatic glands above mentioned, showing much fibrous stroma.

3. CHANGES IN THE LIVER, KIDNEYS, &c.—Similar lymphoid growths may appear, though they do so more rarely in the liver and other organs. They have also been described as occurring in the skin.

4. CHANGES IN THE BONE MARROW.—A similar hyperplasia of the lymphoid cells is seen irregularly throughout the marrow, reminding us of the changes seen in lymphatic leukæmia.

5. CHANGES IN THE BLOOD.—There is little or no change in the early stages of the disease. It is only later (after the disease has lasted some time) that a distinct anæmia is seen, and even then it is not profound except in rapid cases, or in the grave later stages of the chronic cases. The red cells rarely fall below 50 per cent. of the number when the disease is fully pronounced, though later, when it becomes more grave, the number may fall much lower. The hæmoglobin falls disproportionately at first, so that the colour index is low, but later, as the loss in red cells becomes more marked, the colour index rises. Nucleated red cells are usually very scanty. The leucocytes in typical cases are not changed, there being no leucocytosis, thus contrasting strongly with leukæmia; but in the later stages of the disease a distinct leucocytosis (which is generally due to an increase of the polymorphonuclear forms, indicating probably a hæmorrhage, or much fever, or some intercurrent infection) may occur. Occasionally, however, the leucocytosis is due to an increase of the lymphocytes, or there is the appearance of a lymphocytosis, either relative or actual, and when the nature of the change within the lymph-glands is considered, it is surprising that this lymphocytosis is not more commonly seen. In the occasional cases in which this is marked, it may be difficult or impossible to distinguish between this disease and lymphatic leukæmia.

Symptoms.—The enlargement of the glands is the first and most important symptom. It is progressive, though long intervals of quiescence or even of retrocession may arise. The enlargement is painless, but in the later stages, after it has become considerable, it may give rise to

various pressure symptoms, of which pain may be one. Anæmia becomes sooner or later a striking feature in the case, and attacks of hæmorrhage from the nose or elsewhere occasionally occur. Shortness of breath is usually due to anæmia, but may be due to pressure upon the trachea or bronchi. Pyrexia is an important and almost constant symptom. It usually lasts for some time, and occurs after varying intervals, or it may be continuous or show evening rises. There may be pigmentation of the skin. Usually emaciation becomes marked, and gradually the patient loses strength and dies from exhaustion or inter-current disease.

Diagnosis.—There is no difficulty in typical cases, but in others it may be, as already explained, very difficult or impossible to distinguish this disease from tuberculous adenitis, lymphosarcoma, or lymphatic leukæmia.

Prognosis.—Many cases last for years, but the ultimate course is generally downwards. Great improvement often occurs, and the enlarged glands may almost regain their normal size, but it has never yet been proved that they actually become normal again, and it is doubted if a true recovery ever does take place, though evidence on this point is imperfect.

Treatment.—The usual recommendation to remove at once all glands affected at an early period in the disease is good, but the diagnosis at that time is often uncertain. Arsenic is the chief remedy, and in a few recorded cases has proved most successful. It should be pushed short of poisoning. Phosphorus, cod-liver oil, iron, and quinine are worth a trial. Symptoms may call for special treatment.

SUB-GROUP (β)—*THE HÆMORRHAGIC DISEASES.*

This is a group of diseases in which the occurrence of hæmorrhages is a most striking feature; whereas in many of the diseases we have so far considered, hæmorrhages, though sometimes frequent, do not bulk nearly so largely in their clinical picture. In all diseases in which hæmorrhages are frequent or considerable, anæmia becomes a prominent feature, but its relationship to the hæmorrhages is not always the same. In those diseases hitherto considered, the anæmia, in most cases at any rate, precedes the hæmorrhage, and may fairly be blamed for it, and certainly aggravates it, probably by causing some degenerative change in the vessel walls; but in the so-called hæmorrhagic group of blood diseases the anæmia is subsequent to the hæmorrhage, and is probably caused by it. The diseases generally included in this group are—Hæmophilia, Purpura in its various forms, and Scurvy; while Hæmoglobinuria has many claims to be included.

HÆMOPHILIA.

Etiology.—This is a rare affection, showing, like colour blindness and pseudo-hypertrophic paralysis, a remarkable hereditary relationship. It affects males almost exclusively; but while the females themselves generally escape, they transmit the proclivity to their male progeny. In most it appears early in life, and consists in the occurrence of

frequent and often persistent and severe hæmorrhages, either after some trivial injury or spontaneously. The victims are often referred to as "bleeders." The blood, the blood-vessels, and the nervous system have all been blamed, but definite knowledge is still wanting. The proclivity tends to die out in a few, but may exceptionally persist for many, generations.

Symptoms.—A trivial blow or wound or the pulling of a tooth may set up hæmorrhage, which persists in spite of all remedies; or the hæmorrhage may arise spontaneously, the nose being the commonest site, but it may be the skin, mucous membrane, serous surface, &c. Joint affections, especially the larger ones, such as the knee, are very common, and may be due to inflammatory attacks or to hæmorrhage. Bleeders are generally healthy-looking individuals until anæmia is set up by the blood losses.

Diagnosis.—The family history and the recurrent attacks of hæmorrhage—uncontrollable, or controllable with difficulty—generally make the diagnosis easy.

Prognosis.—Bleeders generally die in early youth, and, generally speaking, the younger the proclivity shows itself, the more serious it is. They rarely die from the first hæmorrhage. They may with great care reach boyhood or manhood, or even old age, and the older they get, the weaker the tendency becomes, until it generally, though not always, dies out.

Treatment.—Carefully guard "bleeders" from injuries, and above all from operations, however trivial. When a hæmorrhage occurs compression is most reliable, and local styptics should be employed, especially adrenalin; while ergot, chloride of calcium, or big doses of the tincture of the perchloride of iron may be administered internally. Ice

may be applied locally, and gelatin painted on in a 5 to 10% solution is good. . Where epistaxis is present the posterior and anterior nares generally require to be plugged. Much care during convalescence is essential. Transmission being through the female line, the daughters of a bleeder's family ought not to marry.

PURPURA.

Like many another term in medicine, purpura was originally employed to signify a special feature of a disease or diseases, viz., the occurrence of skin hæmorrhages, and thus was even in its earliest use little better than a makeshift. Now that extended knowledge has shown that a very large number of different diseases have this character in common, its imperfection has become still more obvious, and any classification of such purpuric diseases is necessarily open to serious objection. For the sake of convenience, the term is still in use and a classification attempted. The following classification is fairly complete—(1) Symptomatic; (2) Arthritic; and (3) Hæmorrhagic. The blood escapes by rupture, or by diapedesis, or it is asserted that there may be simply transudation of colouring matter. The extravasation is usually into the upper layers of the corium, sometimes extending upwards into the epidermis, and downwards into the subcutaneous tissue.

1. SYMPTOMATIC PURPURA.—This name may be given to the large class in which skin hæmorrhages of spontaneous origin form a more or less prominent symptom of the chief disease. The origin of the hæmorrhages is not always the same, and we may distinguish—(1) Infectious; (2) toxic; (3) cachectic; (4) mechanical; and (5) neurotic forms. (1) *The infectious forms.*—In many severe infections

—e.g., pyæmia, septicæmia, and ulcerative endocarditis—small cutaneous hæmorrhages are common. This is true also of many of the infectious fevers, notably typhus, many cases of small-pox, measles, scarlet fever, &c. (2) *The toxic forms*.—Group 1. is really also toxic, but it is placed in a special group to show that the toxin is of bacterial production, whereas in this group, poisons of non-bacterial production are referred to, such as snake bite, drugs, jaundice, anæmia, &c. The two groups may run into one another in some cases, as perhaps in jaundice, &c. (3) *The cachectic forms*.—This group might be merged in the last two, as the hæmorrhages met with therein are probably either of bacterial or toxic production. Bright's disease, scurvy, and cancer are examples. (4) *The mechanical forms*.—These are seen in cases of mechanical obstruction to the circulation, as in asphyxia, and in venous congestions generally. (5) *The neurotic forms*.—These are met with in certain rare forms of hysteria, as in the "stigmata" of religious fanatics, in certain reflex disturbances, certain mental shocks, and also rarely in certain organic diseases, such as locomotor ataxia and acute myelitis.

2. ARTHRITIC PURPURA.—Associated with joint trouble and sometimes called rheumatic. Three forms are recognised—(1) Simplex; (2) rheumatic; and (3) Henoch's. (1) *Purpura simplex* is a mild affection, most commonly seen in children. There is some malaise, with or without joint pain. Small scattered bright hæmorrhagic spots appear upon the ankles and legs, less commonly elsewhere, and fade in two or three days. Successive crops may appear, and there may be associated some diarrhœa and slight anæmia. Recovery generally occurs in about ten days. (2) *Purpura rheumatica*, often also called peliosis rheumatica, is a more severe disease, with more marked rheumatic manifestations. There is generally painful swelling of several joints, sore throat, and some fever, and

it occurs generally at a later period than the simple form, viz., between 20 and 30 years of age, and is most common in males. The purpuric eruption is mainly seen on the lower limbs, particularly in front of the legs and around joints. It may be in the form of small hæmorrhages, or urticarial wheals, or nodular infiltrations like those of erythema nodosum, or much more rarely as small vesicles (pemphigoid purpura). Relapses are very common. (3) *Henoch's purpura*.—This is a form described by Henoch and seen chiefly in children in which gastro-intestinal attacks are prominent, viz., pain, vomiting, and diarrhœa. It otherwise resembles the last form, except that hæmorrhages from the mucous membranes or kidney are often very severe, while the joint affections are often slight. Relapses are very common, and may extend over several years. The prognosis is not so favourable.

3. HÆMORRHAGIC PURPURA. — Morbus maculosis of Werlhof. This is the most severe form of purpura, and has come by custom to be called by the inapt term "hæmorrhagic." It is most commonly seen in youth and early adult life, and particularly in girls. Hæmorrhages appear on the skin all over the body and rapidly increase in numbers, and may run together. Bleeding from the mucous membranes (epistaxis, hæmatemesis, hæmoptysis, hæmaturia, melæna) may set in at the same time or later and cause anæmia or death, but death may occur without any bleeding from a mucous membrane. There is usually slight fever. In favourable cases the disease usually subsides in about a fortnight, but it may leave a marked anæmia behind. In the most severe forms (purpura fulminans) the hæmorrhages may be so great that death occurs in from 24 hours to a few days. Many of the cases of purpura hæmorrhagica suggest an infectious origin, like cases of hæmorrhagic small-pox, and future investigation may demonstrate this; but other cases are more allied to hæmophilia, from which they are not easily distinguished.

Treatment.—Where purpura is symptomatic, special attention should be paid to the patient's general condition, and every effort made to support the strength. In purpura simplex, rest in bed, tonics, careful dieting, and the administration of arsenic are helpful. Where there is association with rheumatism give salicylates.

Purpura hæmorrhagica requires vigorous treatment for the arrest of hæmorrhage. Perhaps oil of turpentine (10 to 20 minims), chloride of calcium (20 grains), and some preparation of the suprarenal gland are amongst the best styptics. Absolute rest and careful dieting are most important. In Henoch's purpura an icebag applied over the abdomen affords much relief.

SCURVY.

Syn. *Scorbutus*.

DEFINITION—A disease characterised by a tendency to hæmorrhage and by anæmia, also by a spongy condition of the gums and great weakness.

Etiology.—An improper diet has long been held to be the cause of scurvy. Too much flesh meat, especially if salted, and an absence of fresh vegetables are generally regarded as the main dietetic defects in the causation of the disease, but it has not been determined which particular chemical constituents are at fault in such a defective diet. It is remarkable how slight a change of diet will stop an outbreak, thus a mere change in the water-supply has been followed by the disappearance of an epidemic among troops. Some authorities regard the dietetic error as merely predisposing to the disease, and seek for the real cause

among the bacteria ; but evidence in support of this view is almost completely wanting. The latest theory regards it as produced by ptomaines, which are so common in certain tinned and preserved meats, and hence the want of fresh meat, and not of vegetables, is looked upon as the all-important factor.

Pathology.—Hæmorrhages are very characteristic features. They occur in the skin (where they are generally small), in the subcutaneous tissues, and into the muscles (where they are often large), and in the internal organs, particularly the nose, stomach, intestines and kidneys, and in all the serous membranes. The gums are swollen, spongy, and sometimes ulcerated. The blood changes are those of a secondary anæmia from hæmorrhage. A distinct leucocytosis is often present where the hæmorrhage or some inflammatory complication becomes pronounced. Cloudy swelling is seen in the glandular organs, particularly the liver and kidney, and also in the heart.

Symptoms.—The sufferer is generally pale, with a sallow earthy complexion. There is gradual emaciation, with debility. The hæmorrhages occur later and may form irregular, purplish patches or firm, brawny swellings, especially in the buttocks, thighs and calves of the legs. The spongy fungating gums bleed readily; indeed, the slightest bruise on any part of the body is apt to be followed by more or less extensive hæmorrhage. The general weakness increases, all exertion is irksome, and headache, breathlessness, oedema of ankles, &c., may, as in all anæmias, become marked. Attacks of syncope may come on. Night blindness may occur. Bleeding from the nose is the most frequent of the mucous hæmorrhages, but in severe cases they may occur also from the bowel, &c. There is rarely any fever. Diarrhœa, pneumonia and other complications may occur.

Prognosis.—In favourable cases a simple change of diet may suffice, and in all cases the sooner the dietetic error is corrected the quicker is the recovery. Most cases in which appropriate treatment is used early enough recover completely, though adhesions about the joints and contractions in the muscles may remain. Very severe cases may prove rapidly fatal.

Treatment.—Give fresh meat and vegetables, and often in a severe case they must be administered in easily assimilated form and in very small quantities at a time. Lime or lemon juice should be freely used, and a tonic containing iron is necessary. Treat the local condition of the mouth with chlorate of potash, permanganate of potash, listerine or other antiseptic mouth-wash. In a very severe case painting the spongy gums with dilute silver nitrate solution greatly expedites recovery. Where there are severe hæmorrhages, styptics are needed. Rest is of first importance after supplying the deficient dietetic remedies.

INFANTILE SCURVY.

Hand-fed infants are liable to this disease between the ages of six and eighteen months. Swellings along the shafts of the long bones, particularly the femur and tibia, become noticeable. They are painful, and the child is fretful and cries when touched. The swellings have a pyriform shape, and are most prominent over the epiphyseal cartilages. They are due to hæmorrhages beneath the periosteum and endosteum, and thus resemble rickets. These hæmorrhages occur especially in the areas of the greatest physiological activity, such as the inner layer of

the periosteum and epiphyseal lines. The bone beneath the hæmorrhages may be partly absorbed and replaced by a new and more spongy bone, and bone formation does not occur in the proliferating epiphyseal cartilages, so that the epiphyses separate easily. There are very often other signs of rickets, and this disease was known as acute rickets until Sir Thomas Barlow showed that it was really scurvy. The attitude of the child is noteworthy. It lies fairly quiet when left alone, with its legs drawn up and still. Any movement of its legs causes pain. After the swellings in the legs become marked, the legs, instead of being flexed, lie everted and flaccid, as if paralysed. The change spreads to the upper extremities. The sternum, with its adjacent costal cartilages, and ribs seem to have sunk bodily backwards. Muscular hæmorrhages are much rarer than in adults.

Treatment.—Give fresh milk, small quantities of raw meat juice or potato cream. In some cases lemon juice may be used with advantage.

SUB-GROUP (γ)—*SPECIAL CONDITIONS IN
WHICH RED BLOOD-CELLS ARE
RAPIDLY DESTROYED.*

BURNS.

Many of the red cells have been split up and rapidly destroyed in some fatal cases of general burns, leading to a solution of hæmoglobin in the blood and its presence in the urine.

POISONS.

The same thing occurs after certain poisons—*e.g.*, cobra poison, arseniuretted hydrogen, guaiacol and nitrites—while others not only dissolve red cells but at once transform the hæmoglobin into methæmoglobin, giving the blood and urine a dark-red, almost chocolate, colour—*e.g.*, potassium chlorate and antifebrin. A form due to some unknown toxic agent is seen in horses which after being in the stable for several days are taken out and driven, particularly in cold weather. It comes on suddenly, and there is paresis of the hind legs as well as the dark-coloured urine. Death may occur in a few hours or days. Carbon monoxide gives the blood a cherry-red colour, due to CO.H_6 . Hydrocyanic acid also causes a bright-red colour, due to cyan-methæmoglobin; but clinically the most striking and important condition in which there is rapid solution of red cells in the blood is known as Paroxysmal Hæmoglobinuria.

PAROXYSMAL HÆMOGLOBINURIA.

The older name hæmatinuria is not correct, for the urine contains hæmoglobin or methæmoglobin, not hæmatin.

Etiology.—It is more common in males than females, and appears oftener after than before puberty. Exposure to cold, fatigue, or mental excitement may bring on an attack. The apparent cause may be very trivial in susceptible persons, *e.g.*, dipping the hands in cold water. Some cases seem to be related to either Raynaud's disease, malaria, syphilis, or rheumatism.

Symptoms.—There is generally some disturbance, such as headache, nausea, vomiting, diarrhoea, or pains in the loins, and then the patient passes urine altered in a remarkable way. It has a deep red or reddish-brown colour, often like port wine or porter. It contains albumen but no red blood-cells, though it gives the hæmoglobin reaction with guaiacum and ozonic ether, and the spectroscope shows the presence of methæmoglobin, with occasionally oxyhæmoglobin also. There may be no other symptoms, and the attack may never recur, but usually it does so after varying intervals of time, especially on exposure to cold or other provocative, and susceptible individuals never seem to lose the tendency. It does not seem to shorten life, and cases are rarely, if ever, fatal. For the relations of hæmoglobinuria to blackwater fever, see page 248.

Treatment.—During the attack keep the patient warm and give hot drinks. Nitrite of amyl sometimes cuts short an attack. Avoid cold should it seem to predispose, and treat any disease, such as malaria, syphilis or rheumatism, with which the hæmoglobinuria may seem to be associated.

III.—DISEASES OF THE DUCTLESS GLANDS.

THE DUCTLESS GLANDS.

The thyroid, the suprarenals, the thymus and the pituitary body are called the ductless glands. They have no external secretion or excretion, but they (at least in the case of the first two, and probably also in the others) elaborate certain substances which pass into the general circulation by the blood- or lymph-channels, or both, and exert an important influence upon the nutrition of the

organs and tissues of the body. This function is spoken of as an internal secretion. Diseases of these glands reach their chief importance by producing disturbances in this secretion. Thus two important diseases are directly related to disturbances in the thyroid secretion, viz., myxœdema, in which it is diminished, and exophthalmic goitre, in which it is increased.

SUB-GROUP (a)—*THE THYROID GLAND.*

The colloid substance contained within the saccules of the thyroid gland is elaborated from the blood by the activity of the cubico-columnar cells lining the saccules. It is a complex proteid substance containing a considerable amount of iodine and some phosphorus. It can be broken up into a proteid body containing a small amount of iodine and a non-proteid body containing a large amount of iodine. This latter substance, known as thyriodine, is by far the most active constituent of the colloid substance, and recent chemical researches have shown that the amount of iodine in the thyroids of the inhabitants of a district varies inversely with the prevalence of goitre therein. There is some evidence to show that the colloid substance of the thyroid gland leaves it and reaches the general circulation by way of the lymphatics, though this is not indubitably proved. It is carried by the blood to the tissues and organs of the body, particularly the nervous system, and exerts a most important action upon their healthy metabolism, whether as a trophic substance necessary for their healthy nutrition or as an antitoxic substance neutralising some toxic product of healthy metabolism is not yet known. In addition to the thyroid there exists in man and many other animals another tissue of a different nature, contiguous to the thyroid. It consists of rows of

cells compactly arranged in a network or fairly regular lobules, without, however, any alveoli containing colloid material, but having numerous blood-vessels lying in a scanty interstitial tissue. They are called the parathyroid glands. In man and in the monkey they lie scattered in the capsule and throughout the substance of the thyroid body, in the dog they are in masses, usually situated on its anterior and posterior surfaces, and in the rabbit on each side of the trachea below, and separated from the thyroid itself. These parathyroids are quite distinct from accessory thyroid glands, which are often to be found, both in man and other animals, near the thyroid—for instance, behind the manubrium sterni. The parathyroids and thyroid together are essential to life, for their complete removal is followed by death in all animals. The influence of the former appears to be even more vital than that of the latter, as their complete removal alone usually causes death though the thyroid be left, while, on the other hand, complete removal of the thyroids without the parathyroids, as in the rabbit, does not cause death. The close connection between these glands is further shown by the fact that partial removals of either thyroid or parathyroid tissue are followed by hypertrophic changes within the thyroid, but the new tissue is structurally like parathyroid and not thyroidal tissue. Partial removals of the thyroid gland (necessarily of parathyroid also) in monkeys, when performed very gradually, are followed in time by changes characteristic of myxœdema, viz., loss of hair, dry skin, swelling of subcutaneous tissues, mental failure and emaciation, and unilateral removal of the parathyroid, along with slighter removals of thyroid tissue in dogs, have shown a tendency to produce an exophthalmos, but an enophthalmos when accompanied by large removals of thyroid tissue. There is also some evidence suggesting that the pituitary body may play a part, as it has been shown to enlarge after thyroidectomy, but the active principle of its extract is

somewhat antagonistic to that of the thyroid. The relationships between these three glands have not yet been further determined, but the artificial administration of thyroid extract naturally suggested by these experiments (whether hypodermically or by the mouth is immaterial) has given strikingly beneficial results in diseased conditions dependent upon a diminished natural thyroid secretion, as in myxœdema and cretinism.

DISEASES OF THE THYROID GLAND.

INFLAMMATION.—Inflammation is more frequent in goitrous than in healthy thyroids, in which it is rare. It may be simple, suppurative, tuberculous or syphilitic.

GOITRE.

This term, or its synonym bronchocele or struma, is used to denote all forms of enlargement of the thyroid. This enlargement may be unaccompanied by other signs of disease, all the trouble arising from the mechanical results of the enlargement itself. This is the case in what is sometimes called ordinary goitre—a congenital, or, as is more frequent, an acquired, condition. It is called *parenchymatous* when the number of vesicles is increased, *colloid* when some of these are unduly dilated, and *vascular* when there is a formation of new or dilatation of old vessels in the stroma. Further changes of a secondary nature may subsequently occur, leading to fibrous thickening of the interstitial tissue or dilatation into cysts of some of the saccules, with alteration in their contents. Both the interstitial tissue and the contents of the saccules may become calcified or permeated with blood, and other

changes, such as fatty and waxy degeneration, may be seen. The goitre may cause the patient no trouble beyond the deformity, or it may lead to serious pressure upon the trachea, the vessels, or the nerves, particularly the recurrent laryngeal. The cause of the enlargement is obscure. It is endemic in certain districts—Switzerland, Savoy, Tyrol—and sporadic cases occur everywhere. It is more common in women than men, and generally begins about the period of puberty. There is much evidence to show that the drinking water is to blame, and that it probably derives its causative influence from certain constituents of the soil. It has been recently asserted that the medicinal use of thyroid extract causes a marked diminution in the size of these goitres, thus suggesting that they owe their origin to an increased demand for thyroid secretion. Other goitres are associated with an undoubted interference with thyroid secretions, either diminishing or suspending it, with a consequent development of myxœdema or cretinism, or increasing it as in exophthalmic goitre. These are general diseases, and not merely local, as may be the case in other goitres.

Treatment.—As indicated above, thyroid extract is beneficial in some cases; while ergot and potassium iodide have been found useful. Iodine externally might be tried. If possible remove the patient to a district free from goitre. In certain cases surgical interference may be resorted to.

MYXŒDEMA.

It is a very chronic disease of adult life, ten times more common in women than men, due to reduction or even suspension of the thyroid secretion. The thyroid gland is

be smaller or absent, as in most of the sporadic cases. It usually begins from the second to the fifth year, and is rarely congenital. It chiefly affects females. The arrest of growth is so great that the individuals, though old in years, retain their childlike appearance. The skin is thick, the abdomen is large and pendulous, the limbs small but the hands, feet and head large. The basisphenoid and basioccipital unite early, instead of from the fifteenth to the twentieth year, and the great size of the epiphyses of the long bones and the short size of their shafts are due to a large growth of periosteal bone at the epiphyseal junctions. It grows inwards, and may entirely shut off the epiphyses from the shaft. There may be complete absence of speech and all intelligence, or these may be merely defective. These different degrees have given rise to the terms semi-cretinism and the cretinoid state.

Treatment.—Thyroid extract must be cautiously administered, and in a successful case will be followed by both physical and mental development.

EXOPHTHALMIC GOITRE.

Syn. Graves' Disease, Basedow's Disease.

DEFINITION—A chronic disease, chiefly met with in young adult women, very rare in men, characterised by enlargement of the thyroid, protrusion of the eyeballs, palpitation, and nervousness.

Etiology.—It has frequently been thought to follow upon a mental shock, fright, overwork, or worry. Associations have also been noted with other conditions, but the cause remains obscure.

Morbid Anatomy.—The thyroid gland is usually considerably enlarged, either symmetrically or the right lobe predominating. It is soft or firm and sometimes nodular. Accessory thyroids, if present, may be similarly affected, and the thymus persists and is enlarged as in Addison's disease, lymphadenoma and some cases of leukæmia. Microscopically, the alveoli are opened out and papilliferous, and are lined by a columnar epithelium and contain no increase of colloid material, sometimes indeed little or none, or it is replaced by a mucoid-like fluid. This is the structure of normal parathyroid tissue, and is similar, as already mentioned, to that produced within the thyroid after partial thyroidectomy. The stroma shows a moderate diffuse round-celled infiltration which may result in much fibroid interstitial change and atrophy of the epithelium. There may also be some enlargement of the heart, spleen and lymphatic glands.

Pathology.—Both an increase in quantity and an alteration in quality of the thyroid secretion are thought to occur in this disease, and this appears to receive corroboration from operations tending to lessen its secretion. Partial thyroidectomy or diminution of the blood-supply to large parts of the gland usually lead to improvement, and total thyroidectomy may be followed by complete cure. On the other hand, the symptoms do not vary in severity with the degree of enlargement of the gland, which ought to occur if this hypothesis be correct, so that we cannot yet accept it without doubt.

Symptoms.—There are three cardinal symptoms—the goitre, the protrusion of the eyeballs (proptosis or exophthalmos) and palpitation. They vary greatly in different cases, the last being usually the most constant of the three, and usually the first to be complained of.

THE GOITRE.—This is usually symmetrical, and varies much in degree in different cases and at different times in the same case. It may give visible pulsation, a palpable thrill, and an audible limit.

THE EXOPHTHALMOS.—This also is symmetrical, though usually greater in the one eye than in the other. It may be slight or so marked that the eyelids cannot voluntarily be closed. There is retraction of the upper eyelid, due to spasm of the levator palpebræ, so that the palpebral fissure is enlarged, and a ring of sclerotic is seen all round the cornea and there is diminished frequency of winking under reflex stimulation. This is known as Stellwag's sign. Another sign of great diagnostic importance is Von Graefe's sign. It is generally present, being well-marked even in cases where the exophthalmos is slight in amount. It consists in the upper eyelids not keeping pace with but lagging behind the eyeballs in their downward movement, so that the sclerotic above the cornea may become visible. It is sometimes observed in other conditions. Inflammation of the conjunctiva or ulceration of the cornea is rare, even when the exposure is great.

THE PALPITATION.—This is generally the chief trouble complained of. The heart beat is regular but rapid, about 100 in slight cases, 120 in moderate, and up to 160 or more in severe cases. Both the palpitation and the tachycardia are not merely persistent, but apt to become extreme on the slightest exertion or excitement. Besides these three main symptoms, there are others more or less constantly met with, such as tremor, emaciation and loss of strength, pigmentation of the skin, dyspnœa, extreme nervousness and other nervous phenomena. Tremor is the most important of these. It is of a fine character, having a small amplitude with a period of about one-eighth or one-ninth of a second, like the senile or alcoholic tremor. It is best seen in the limbs, particularly the arms and hands, and is exaggerated on exertion or excitement. Emaciation

is seen in all acute cases, or when the disease is active. Pigmentation of the skin is not uncommon. The skin of the face and other parts, such as the nipples, abdomen, flexures of the arms, &c., becomes muddy-looking, or of a more or less brown colour in patches. The hair often becomes thin and dry. Dyspnœa may be very severe and even prove fatal. It is paroxysmal and is accompanied by severe palpitation. There is generally great nervousness, and often irritability or depression and insomnia.

Diagnosis.—It is easy when exophthalmos and goitre are present, and when they are absent the tremor, palpitation and tachycardia are the best guides.

Prognosis.—The disease may become arrested or improved, or even cured; on the other hand, it is often fatal, sometimes suddenly so, death being due to asthenia, dyspnœa, or the onset of mania or vomiting. It is sometimes followed by myxœdema, or much less frequently by diabetes mellitus.

Treatment.—There is no stereotyped treatment for this disease, and different cases require very different methods. In the first place, insist upon complete rest for a prolonged period, with a wholesome dietary and freedom from all excitement. Try phosphate of soda in one drachm doses thrice daily for at least one to two months. Anti-thyroidin is worth a trial, but has yielded disappointing results in our hands. Sedatives, such as belladonna and bromides, are often beneficial. Strophanthus or digitalis is often necessary for the cardiac condition, and iron and general tonics may be administered in nearly every case. Ergot has been recommended, but is disappointing. Galvanism and faradism applied in different ways are often tried but are generally useless. Operative treatment is indicated in cases which are not too far advanced, and in which

all else has failed. Ligaturing the isthmus and removal of a lobe, or even the whole gland, have been performed, but the death-rate is uncomfortably high. Excision of the cervical sympathetic ganglia only removes the exophthalmos but does not relieve the other symptoms. An ice-bag applied for days at a time over the heart or neck, or both, will be found of very great benefit.

TUMORS OF THE THYROID.

Simple tumors are rare, and include fibroma, chondroma, osteoma and adenoma. The latter is the most interesting. It forms an isolated mass within the thyroid, which it resembles in structure. It is difficult to distinguish between these adenomas and nodular goitrous enlargements. Malignant tumors are more frequent, several varieties of both sarcoma and cancer being met with. The most interesting is the adeno-carcinoma, which consists of masses of rounded or oval epithelial cells in a vascular stroma. Colloid changes are often seen in the cells, and in the early stages a regular alveolar arrangement containing colloid, like the normal thyroid, may occur. They form nodular or diffuse infiltrations in both goitrous and normal glands, and they infiltrate the surrounding tissues and cause metastasis, particularly in the lungs and bone. They may or may not cause a visible enlargement of the gland.

Treatment.—The treatment is mainly surgical. In certain cases where the gland is completely destroyed, thyroid extract should be administered.

SUB GROUP (β)—*THE SUPRARENAL OR
ADRENAL GLAND.*

Complete removal of all suprarenal tissue is followed in all animals by death, and their blood possesses definite toxic properties which are neutralised by the addition of adrenal extract. The most remarkable property of adrenal extract is its action upon muscle. It is a muscular tonic and stimulant, and therefore causes a marked, though transient, rise of blood-pressure by contracting the arterioles. This explains why disease or removal of the adrenals is followed by asthenia and low blood-pressure. Adrenaline, which is now extensively used as an astringent, is a very active crystalline substance obtained from adrenal extract. The extract has other actions. It slows the heart beat, probably by stimulating the cardio-inhibitory centre in the medulla. It is elaborated within the medullary portion of the glands and secreted into the blood of the adrenal vein. Its administration by the mouth has no effect.

DISEASES OF THE SUPRARENAL BODY.

MALFORMATIONS of various kinds occur. Of these the most important is the presence of accessory glands—so-called adrenal rests. They are frequent and found oftenest in the connective tissue around the chief adrenal body; but they may be present in the kidneys, the right lobe of the liver, along the renal vessels, and in other and more distant situations—*e.g.*, in the broad ligaments. Their chief importance lies in the possibility of their enlargement to compensate for a diminution or destruction of the

function of the main glands, and also in their being the seat of origin of adrenal tumors.

DEGENERATIONS.—The ordinary forms occur, but call for no special mention.

HÆMORRHAGES.—They may be large and rupture the gland, or be small. They are most frequent in the medulla, and are of chief importance in new-born children (where they occur chiefly in difficult labours), owing to the subsequent cicatrisation following upon their absorption ultimately producing a diminution in the internal secretion of the gland.

ADDISON'S DISEASE.

DEFINITION—A chronic disease chiefly affecting males, characterised by gastro-intestinal disturbance (vomiting and diarrhœa), extreme asthenia, bradycardia, and pigmentation of the skin and mucous membranes.

Etiology.—It chiefly affects adult males between thirty and forty years of age, but it is not uncommon in females. Severe mental strain and injuries to the abdomen and back have been blamed for its onset, but belief now mainly centres in a diminution or cessation of the internal secretion of the gland as the immediate determining cause, and any diseased condition which brings this about may be regarded as the primary cause of the disorder.

Morbid Anatomy.—In the great majority of cases a fibro-caseous tuberculosis of the gland, which begins in and is most marked in the medulla, has been present. It is usually bilateral, but occasionally unilateral. The fibrosis around the caseous masses extends beyond the capsule of the gland to the surrounding structures, often leading to a

matting together of the whole, which may involve the sympathetic ganglia and nerves. Tuberculous masses may be present in the suprarenals, along with tuberculosis elsewhere, without causing Addison's disease. They only do so where they seriously interfere with the internal secretion. A simple atrophy of obscure origin is present in other cases. Malignant disease, particularly secondary, may be the cause in exceptional cases; but in the majority of cases of hitherto reported destruction of the adrenal glands by malignant disease, the symptom complex of typical Addison's disease has not been present. In certain cases the adrenal glands themselves have been healthy, but surrounded by extensive chronic fibrous inflammation which might have led to destruction of the efferent vessels of the gland—as effective a means of stopping its internal secretion as destruction of the gland itself.

Pathology.—Addison himself suggested that the disease was due to an alteration in the function or secretion of the adrenal gland, but from the frequency with which the structures around the glands, particularly the sympathetic nerves and ganglia, were found to be involved in the surrounding fibrosis, attention became focussed upon these latter structures, and it was suggested that the disease owed its origin to degenerative and inflammatory changes in these nervous structures. This suggestion appeared to be supported by the cases in which the suprarenal glands themselves were found to be normal. Of late, opinion has again veered round to Addison's view, and the main objection which is urged against it is that the suprarenal glands are not infrequently entirely destroyed by disease, particularly by malignant disease, without Addison's disease supervening. But this objection can be met by the possibility of accessory adrenals having been present in those cases, and, further, by the supposition that when the destruction of the adrenals is rapid, there is not time for

all the symptoms characteristic of Addison's disease to appear. The profound asthenia and the bradycardia are there, but there is not time for the pigmentation to be produced. The former are due to the withdrawal from the blood of the adrenal secretion, which acts as a powerful physiological tonic to all muscular structures. This withdrawal may allow of the gradual development of toxic substances within the blood, which cause in turn the vomiting and diarrhoea, and possibly also the pigmentation, by an action on the sympathetic nervous system; but this is mainly a hypothesis. The pigment lies in the deeper layers of the epithelium of the skin and mucous membranes, and there is clinical evidence in support of the influence exercised by the nervous system upon pigmentation of the skin.

Symptoms.—There is no priority among the three leading symptoms, viz., asthenia, gastro-intestinal irritation and pigmentation, but the most striking of them—the pigmentation—is usually preceded by a feeling of increasing weakness of insidious onset, pains in the loins, and occasional attacks of vomiting. Occasionally the leading symptoms come on together.

1. *Asthenia.*—Weakness is a striking and characteristic feature of the disease. There is an early feeling of tiredness, which goes on increasing without any obvious cause. The prostration of strength in time becomes very marked, although the muscles may be firm and show no sign of being wasted. The muscular weakness affects the heart as well as the voluntary muscles. The pulse is soft and feeble, and the action of the heart slow and irregular. Headache is frequent. Vertigo and syncope, which may prove fatal, may supervene at any time.

2. *The Gastro-intestinal Symptoms.*—There is often complete loss of appetite, fitful attacks of vomiting, or diarrhoea.

3. *Pigmentation*.—It is present in the skin and mucous membranes. In the skin it is diffuse, though most marked where the normal pigment is greatest, as the areolæ of the nipples, the genitals, the groin, or any part subjected to pressure, as by a garter or belt. The tint varies from a yellowish to a deep brown. In the mucous membranes it is patchy. This is best seen in the inside of the lips, cheeks and on the tongue. Pigmentation is said to be absent in a few cases—*i.e.*, there have been cases which had the other symptoms and which, despite the absence of pigmentation, were thought to be examples of Addison's disease.

Diagnosis.—It is difficult in the early stages. Asthenia and pigmentation together justify a diagnosis; but pigmentation alone is apt to mislead, as it is seen in many other diseases, and in vagabonds.

Prognosis.—The disease is usually fatal, the average duration being one to two years, but some cases are very acute and others run a chronic course of several years' duration. The disease may recede and great improvement (in the strength especially) may be seen for many months, but relapses are apt to occur in such cases at any time. Recovery has been reported.

Treatment.—The treatment of Addison's disease falls under two heads—first, the alleviation of the distressing symptoms and, second, an attempt at the artificial replacement of the arrested functions of the gland. The debility should be treated with arsenic, strychnin, iron, and other tonics, but avoiding any remedy which predisposes in the case under observation to either gastric disturbance or diarrhœa. Give bismuth with hydrocyanic acid for the vomiting, and bismuth in large doses for the diarrhœa, and pay special attention to the dietary of the patient.

Suprarenal extract has not been very satisfactory. It is generally administered in tabloid form, each grain of the dry extract being equivalent to 15 grains of the fresh gland, and one tabloid is given thrice daily with care. Only a few patients are actually benefited, while disagreeable consequences of the extract are not uncommon.

TUMORS OF THE SUPRARENALS.

Of the simple tumors, the adenoma is the commonest. Primary and secondary sarcoma and carcinoma are not uncommon. They may destroy the whole glands and affect the tissues around without giving rise to pigmentation of the skin, but there is always intense asthenia.

SUB-GROUP (γ)—THE THYMUS GLAND.

The action of this gland has yet to be determined. Its loss has been followed by asthenia, and the injection into the veins of large doses of its extract by death from dyspnoea. It is large and active during foetal life, and also from birth to puberty, after which it gradually atrophies, until at the end of the twentieth year it is almost completely replaced by fatty tissue. It is found to be enlarged in certain diseases of the blood and of the ductless glands, such as exophthalmic goitre, lymphadenoma and Addison's disease.

DISEASES OF THE THYMUS GLAND.

INFLAMMATIONS, both acute and chronic, occur, particularly in infants, but are not frequent.

HYPERTROPHY.—The gland may enlarge either before or after the period for its normal involution. It has been held by some to be the cause of a paroxysmal form of dyspnoea, known as “thymic asthma” from its pressing directly on the trachea or on the vagi or their inferior laryngeal branches. It is also thought in some way to have been the cause of sudden death from suffocation in otherwise healthy infants as well as in older persons. It has also been frequently found enlarged in exophthalmic goitre, and some of these cases died suddenly.

TUMORS.—Dermoid cysts, sarcomata and carcinomata occur; the first and third arise in epithelial remnants.

Section 4.

LOCAL AND SYSTEMIC DISEASES.

I.—*DISEASES OF THE HEART.*

MALPOSITIONS.

The heart is occasionally found to be situated—(1) Outside the thorax (ectopia cordis), either in the neck, chest or abdomen; (2) inside the thorax, not in its proper position, but either in the middle line or on the right side (dextrocardia). The extrathoracic displacement is usually followed by a short extrauterine life, but sometimes by one of many years. Dextrocardia is usually associated with a similar displacement of the abdominal viscera.

MALFORMATIONS.

These may show themselves in—(1) Defects in the pericardium, (2) the cardiac septa, (3) the orifices and trunks of the large vessels. Pericardial defects are mostly associated with cardiac ones and are not in themselves of clinical importance. Septal defects may be either auricular or ventricular and complete (rare) or partial. The commonest auricular septal defect is a patent foramen ovale. It is only of clinical importance when, as is commonly the

case, it is accompanied by stenosis of the pulmonary artery or deficiency in the interventricular septum. This latter occurs most commonly at the "undefended space," or pars membranacea, near the base of the ventricles. The most important of the arterial malformations are—(1) Stenosis and narrowing of the pulmonary artery or of the aorta, (2) malpositions of these large vessels, and (3) persistent ductus arteriosus. The stenosis may exist at the orifices, when it is due to foetal endocarditis, or the first or further part of the pulmonary artery or aorta, when it may be due to some congenital defect. The malpositions of the large vessels may show an origin of the pulmonary artery from the left ventricle and the aorta, from the right, or both trunks from the same ventricle. A patent ductus arteriosus may exist alone and have little influence upon life; but it is more commonly associated with some other of the defects above mentioned, such as septal defects, stenosis of the pulmonary artery, or a patent foramen ovale.

Symptoms.—The symptoms of all these malpositions are mainly those of dyspnœa, general cyanosis, clubbed fingers, dropsy and various auscultatory murmurs.

Treatment.—Treatment need only be considered in those cases compatible with life. While it must always be remembered that slight malformation may exist without symptoms, and which therefore calls for no interference, the general rules of guidance must be modified to meet the varied symptoms present in each individual case; but rest, exercise, work—physical and mental—and climate are all important. When there is much interference with the circulation, a warm dry climate, warm clothing and special care to avoid the risks of bronchitis, should be taken into consideration and occasional help given to the heart directly by cardiac tonics and indirectly by stimulating the emunctories when such assistance is required.

SUB-GROUP (a)—DISEASES OF PERICARDIUM.

HYDROPERICARDIUM.

A true dropsy of the pericardium may be present in any case of general dropsy, especially renal or cardiac. The fluid measures from a few ounces to a pint or more, and is usually clear, but may be turbid or hæmorrhagic.

Symptoms.—If the amount of fluid be small, the symptoms are slight and generally escape detection. If the amount be large, the existing symptoms caused by the primary disease are aggravated, particularly dyspnœa and cyanosis.

HÆMOPERICARDIUM.

When the fluid is pure blood it comes from the rupture of the heart or an aneurism thereof, or of the aorta, pulmonary artery or coronary arteries; but when it is only mixed with blood it is most likely due to a pericarditis of a malignant, tubercular or other specific origin.

Symptoms.—In the case of pure blood rapidly accumulating within the pericardial sac, collapse and cardiac failure quickly cause death. In the case of a mixed hæmorrhagic and serous or other effusion there may be pain, faintness, syncope or dyspnœa—in short, similar symptoms and signs to those of *grave* pericarditis with effusion.

PNEUMOPERICARDIUM.

Pneumopericardium is so rare that it merely requires mentioning. The gas may be air from without or some

neighbouring air-containing space or a gas generated by bacteria. In either case pyogenic bacteria, and hence pus, are nearly always present and the condition is very grave.

Treatment. — Hydropericardium rarely requires more than the general treatment applicable to dropsical effusions; but if the fluid is causing danger to life, paracentesis (see page 415) may be resorted to. Hæmopericardium rarely permits of any treatment; while in pneumopericardium, when it does occur, the treatment for a purulent effusion should be attempted.

ACUTE PERICARDITIS.

Etiology.—Acute pericarditis may be, but very rarely is, a primary disease. It is primary when arising directly from a wound in the chest involving the pericardium, and perhaps also in some cases of spontaneous pericarditis following after chills, &c. From the analogy of pleurisy these are believed to be mostly tubercular in nature. Pericarditis is thus almost always a secondary disease, though its relationship to the primary disease is not always equally well marked. It is most marked when it extends directly from some neighbouring structure, such as the lung, pleura, myocardium, endocardium, stomach, &c. It is less marked when the infective agent reaches it by the blood-stream, which happens most frequently. This occurs in a great many diseases, *e.g.*, rheumatism, septic conditions, gonorrhoea, scarlet fever, chorea, small-pox, Bright's disease and some constitutional diseases, such as gout and diabetes. The causal organism of the primary disease, when it is known—*e.g.*, the pyogenic cocci, the pneumococcus, the tubercle bacillus—is the cause also of the pericarditis, and is to be found in the inflamed membrane and the effusion. In other cases the circulating toxin present in the primary

disease may weaken the pericardial membranes and render them susceptible to complicating organisms, such as the pyogenic cocci, which may accidentally reach them, or it may be that the toxin itself can set up the inflammation without the subsequent help of bacteria. This is believed by some to be the genesis of some cases of pericarditis following upon Bright's disease. Rheumatism is by far the most frequent of the diseases which cause pericarditis; but whether it does so by toxic or bacterial action or both is not yet ascertained. While certain diseases have been mentioned as generally showing preferably the one or other of these two paths of infection, it must not be concluded that they always do so, for while pneumonia generally causes pericarditis by direct extension, it may do so through the pneumococcus reaching the pericardium by the blood-stream.

Morbid Anatomy.—The appearances are those seen in inflammation of any serous membrane (*vide* "Inflammation"). There is first a general injection of both layers (visceral and parietal), followed by a loss of gloss of the serous surface and then the appearance of a distinct exudate. The amount and character of the exudate are of great importance in determining not only the morbid appearances but some of the chief physical signs. It may vary greatly, not only in different cases but at different times in the same case. It may be scanty or profuse, and serous, sero-fibrinous, fibrinous, purulent or hæmorrhagic, and thus serous, fibrinous, &c., pericarditis are spoken of. In the serous form there is a varying quantity (a few ounces to one or more pints) of a clear yellow or greenish fluid. There is always some fibrinous exudate as well, though it is slight in amount as compared with the serous fluid. It may appear only as a thin gauze-like coating of the serous surface or float in the watery fluid as minute particles or thin threads or largish flakes. This serous variety is most

commonly seen after rheumatism, Bright's disease, tuberculosis or chorea. This exudate may be entirely absorbed and the pericardium regain its normal appearance. In the fibrinous variety there is very little serous, but a varying amount of fibrinous exudate, forming the so-called dry pericarditis. In mild cases it forms a thin gauze- or honeycomb-like layer on the surface of the pericardium, from which it is for a time easily stripped off, disclosing a surface still smooth but generally injected, and often showing minute hæmorrhagic points. In more severe cases the amount of fibrin is greater and forms a thick white or whitish-yellow layer having a rough shaggy surface, aptly likened to the appearance presented by the faces of two thickly buttered slices of bread which have been firmly opposed and then separated. In other cases, the fibrin on the two surfaces is not only shaggy but everywhere drawn out into long threads, giving a hairy-like surface (the hairy heart). The fibrinous exudate is generally thickest about the interventricular grooves and posterior surface, and may be almost limited to these regions; but more commonly it is widely spread all over the heart. This exudate is never completely absorbed, and results in dense white opaque patches in the pericardium (the "milk spots"), or in adhesions between the two layers of the pericardium, as irregular bands or complete adhesion with obliteration of the pericardial sac. In the purulent form there is pus in the sac, either creamy or more watery from admixture with serum. There is generally also some fibrin, either as flakes in the pus or as patches on the heart. It is more common in children than adults. It generally causes death, but recovery is possible when the liquid parts of the pus are absorbed, leaving an amorphous solid mass in which calcification may subsequently occur. In hæmorrhagic pericarditis the exudate, which may be of any of these varieties already mentioned, is mixed with blood in varying quantity. It is seen most commonly in

tubercle and cancer. In pericarditis there are always accompanying changes in the myocardium, *e.g.*, granular and fatty degenerations of the muscular fibres, and some cellular interstitial change. These changes, which are of great practical importance, may be superficial or throughout the whole thickness of the muscle.

Symptoms.—They vary quite as much, if not more, than the morbid appearances. Their gravity, however, generally corresponds to the severity of the morbid changes. Some are general, being due to the absorption of toxins, causing fever (100°F. to 105°F.), thirst, vomiting, dyspnoea, syncope, lividity, delirium, convulsions or coma, according to their severity. The others, and some of the above in part, such as the dyspnoea, syncope and lividity, are due to the mechanical effect of the exudate upon the cardiac contractions and to the accompanying myocarditis. There may be pain over the precordia, and pain on movement or on taking a deep inspiration. Friction may be palpable, but is most frequently heard as a superficial, rough, to and fro sound on auscultation over the precordia, especially over the middle of the sternum and right side of the heart. If there be much serous effusion the area of dulness becomes increased and assumes a pyriform shape, the base being downwards and outwards. The apex of the heart is tilted upwards, but the apex beat may be felt with difficulty or not at all. The lungs may be displaced laterally, and the abdominal viscera downwards.

Diagnosis.—The rough character of the friction sound and its irregular and varying locality (not limited to any of the cardiac valves) and its being usually increased by the pressure of the stethoscope serve to distinguish it from endocarditis. When effusion is present the area of dulness is characteristic.

Prognosis.—This is very grave in the purulent form, but very hopeful in the other forms. The majority of the latter recover, but adhesions of various kinds are a frequent result and may lead to a permanently damaged heart.

Treatment.—The treatment of pericarditis includes also the treatment of the diseases with which it may be associated; and this fact should be borne in mind, for the treatment applicable in acute rheumatism would be obviously dangerous when the pericarditis occurs in typhus, typhoid, &c. Much was hoped from the energetic use of the salicylate group of remedies in checking acute rheumatism and so preventing involvement of endocardium and pericardium. *These hopes have by no means been fulfilled.* Absolute rest in bed where the pericardium is involved, however slightly, and the judicious use of the ice-bag are invaluable. Leeching may help in relieving pain, and small blisters are worth a trial in suitable cases. Opium is invaluable and should be given in sufficient doses to relieve the general and local symptoms. Where an effusion is present, blisters locally, diuretics and purgatives, together with dry diet, will all help absorption. The infusion of digitalis (3j–iv) with the acetate of potash (20 to 30 grains) and magnesium sulphate (3j–iv) are invaluable, and the iodide of potash is useful where the pulse is sufficiently strong.

Paracentesis in the fourth left interspace, close to the sternal border, or through the diaphragm in the angle between the xiphi-sternum and left costal margin, should only be resorted to in extreme cases and a limited amount of fluid withdrawn. If the effusion is purulent and of considerable amount it may be treated as an abscess.

CHRONIC PERICARDITIS.

Etiology.—Chronic pericarditis usually follows upon the acute or subacute disease. Occasionally, however, it arises otherwise in a slow insidious and obscure way. Some of these latter forms are tubercular in origin, others follow upon some of the acute infectious fevers, such as scarlatina and measles, and a good few are difficult to account for.

Morbid Anatomy.—The changes always consist in fibrous tissue formation, which may, however, present a variety of appearances. There may be fibrous bands of varying size and length between the two pericardial surfaces, which are usually themselves somewhat thickened, or the adhesions may be general all over the surfaces, obliterating altogether the pericardial sac (adherent pericardium) or the adhesions may not be confined to the pericardial sac but involve also the outer surface of the parietal layer, causing adhesion to surrounding structures, such as the chest wall, pleura or diaphragm. This form is called indurative mediastino pericarditis or external pericarditis. It is often (more so than the other two varieties) associated with considerable hypertrophy or dilatation of the heart, usually affecting the right more than the left ventricle, occasionally there is atrophy instead.

Symptoms.—In some cases there are practically no symptoms. In others there may be signs of cardiac enlargement, or palpitation, or dyspnœa, or cyanosis or other indication of failure of the right ventricle. A visible retraction of certain parts of the chest wall during ventricular systole is said to occur sometimes, and to form an important diagnostic feature.

Treatment.—Every effort should be made to prevent adhesions forming during the stage of resolution of

pericarditis. Once an adherent pericardium is existent, and if extensive, the symptoms caused may be treated; but little or nothing can be done to remove adhesions already formed. Much will depend upon the extent to which the adhesions interfere with the heart's action and the muscle-fibres of the myocardium. Potassium iodide aids reabsorption, and should at least be tried.

SUB-GROUP (β)—DISEASES OF MYOCARDIUM.

The ordinary degenerations, viz., parenchymatous, fatty and waxy, occur in the myocardium, as in other organs, *vide* page 23.

PARENCHYMATOUS DEGENERATION.

Parenchymatous degeneration occurs in the course of many of the acute infectious diseases, as well as in many cases of pericarditis, and may, besides aggravating some of the symptoms of the primary disease, cause others referable to the impaired muscular power of the heart.

FATTY DEGENERATION.

Fatty degeneration may arise from any of the causes of fatty disease generally, *vide* page 24, or it may arise from certain local causes, the most important of which is stenosis of the coronary arteries. It may show the typical mottling or thrush's breast appearance, generally best seen in the musculi papillares of the left ventricle, or it may be more diffuse, causing no appreciable change in colour, or a

noticeable pallor. The muscle is more flabby and friable, and the ventricular cavities may be dilated. The fat is in the form of longitudinal rows of tiny globules within the muscle-fibres, and it may be, especially when following upon some acute infectious disease, associated with other degenerative sarcoplasmic changes, such as cloudy swelling, vacuolation of the protoplasm, enlargement and vacuolation of the nucleus.

Symptoms.—The onset is usually very gradual. The first symptoms may appear after some unusual effort or spontaneously. Shortness of breath and anginoid pains are most frequent, but there may be palpitation or cerebral symptoms due to deficient blood supply to the brain, such as giddiness, syncope, irritability, loss of memory. Exertion may bring them on at once, while rest may cause them to largely disappear. The pulse is feeble and irregular. The cardiac dulness may be increased and the heart sounds feeble. Sudden death may occur from syncope or rupture of the cardiac muscle.

FATTY INFILTRATION.

Fatty infiltration, usually known as *fatty heart*, differs from the last in there being, at first at any rate, no change in the muscular fibres themselves. The fat is deposited in the epicardium in excess of the normal amount, and while it may be all over the surface of the ventricles, it is most abundant over their bases. Later it invades the inter-muscular septa, even as far as the endocardium, and leads to atrophy of the muscular fibres. It is only exceptionally, and in the later stages, that the muscular fibres themselves show true fatty degeneration. It is most frequently seen in old people of corpulent tendencies, and may exist for a long time before it gives rise to any noticeable symptoms.

Shortness of breath is generally the most marked symptom ; but the others met with in true fatty degeneration may appear, and even sudden death, though not so common as in degeneration, may occur from the same causes.

WAXY DEGENERATION.

Waxy degeneration is fairly frequent, particularly in the right auricle. It occurs in patches, and affects, as elsewhere, the walls of the blood-vessels. It only occurs along with waxy diseases elsewhere.

FIBROID DEGENERATION.

This is a name given to a fibrous tissue formation between the muscular fibres. It may be diffuse, but generally occurs in patches, particularly in the ventricles, and may be large enough to be seen as white or grey streaks or areas. The commonest sites are the wall of the left ventricle near the apex and the apices of the papillary muscles. It is never in itself a true degenerative process, for it occurs only when nutrition to a part of the heart is interfered with, either through a stenosis of the coronary artery or through the deleterious action of some toxin circulating in the blood. In either case the highly specialised muscular fibres degenerate and ultimately atrophy, while the interstitial fibrous tissue undergoes a compensatory hyperplasia. It is most commonly seen as a sequel to some acute process, such as pericarditis or endocarditis, or to the infectious fevers, or to some of the chronic diseases, such as syphilis. The most serious result is when an aneurism, which may lead to rupture, forms at the site of one of these fibrous patches. Cardiac hypertrophy and dilatation sometimes occur.

Symptoms.—They may be absent altogether, and when present they closely resemble those caused by fatty degeneration.

Treatment.—Several of the degenerations above described may be combined to a greater or less extent, and the clinical features are often not sufficiently distinctive to enable the physician to diagnose the exact pathological condition present. Fatty degeneration demands most careful treatment. Try to relieve the over-burdened heart by rest, carefully graduated exercise, *small* doses of cardiac tonics, and a regulated quantity of alcohol. Give small and easily digestible meals, with a limited amount of fluids. Sometimes arsenic, iron and general tonics are beneficial.

Fatty infiltration should be treated by reducing the obesity, of which the condition of the heart forms a most important part. The elimination of fat-forming ingredients from the dietary and greatly diminishing the total fluids ($1\frac{1}{2}$ to 2 pints a day) will, in most cases, rapidly alleviate the symptoms. The Oertel system of graduated hill-climbing and the Nauheim baths and exercises are often of great service.

Fibroid degeneration of the myocardium may cause indefinite symptoms; but if cardiac weakness or dilatation is present, cardiac and general tonics, associated with careful dieting and rest, are the most helpful methods of treatment. Potassium iodide is often useful, and is worth a trial.

SEGMENTATION AND FRAGMENTATION OF MYOCARDIUM.

This condition, to which the name *État Ségementaire* was first given, has received considerable attention of late years. Segmentation refers to separation of the muscle-fibres

along the natural lines of union. It is often found in the heart, mostly the ventricles, after death from suffocation or from some acute infectious disease, and may or may not be associated with degenerative and atrophic changes within the muscle-fibres. Fragmentation refers to the separation of the muscle-fibres into muscle-cells and irregular fragments without reference to their natural cement lines. The significance of these changes is still a matter of doubt.

ATROPHY OF THE MYOCARDIUM.

The heart, like many other organs, is diminished in weight and size in many wasting diseases, notably cancer and diabetes. The atrophy may be accompanied by increased pigmentation, giving the organ a brown colour, when it is spoken of as brown atrophy. It is often said that the heart atrophies in old age, and it doubtless does so in common with other organs, provided that the arteries remain healthy; but when, as is so often the case, the arteries have become somewhat thickened, the heart will generally be found to be a little above the normal size and weight. In the same way, no general statement that the heart atrophies in phthisis should be made, as the increased resistance in the lung may cause both hypertrophy and dilatation of the right side.

MYOCARDITIS.

Acute myocarditis occurs in the outer layers of the myocardium in most cases of acute pericarditis, and in its inner layers in many cases of acute endocarditis; but it may occur independently of these conditions, particularly

in the course of certain acute infectious diseases. It is rarely recognisable by the unaided eye unless it leads to the formation of general or local suppuration (acute suppurative myocarditis), when it shows itself as yellowish-white spots or streaks or obvious abscesses surrounded by congested zones. The abscesses are found unchanged, usually deeply seated, in the substance of the muscle at the base of the left ventricle when death is due to the diseased condition which set up the myocarditis; but in other cases they may rupture into the pericardium and cause purulent pericarditis, or into the heart and cause pyæmic emboli, &c., or establish a communication between the two ventricles when situated in the interventricular septum. They may occasionally dry up and calcify. Whether the inflammation be simple or suppurative, the wall of the left ventricle and the interventricular septum are the parts usually most affected. Even when non-suppurative, the affection is local in the form of leucocyte collections around the dilated blood-vessels and between the muscle-fibres. There is generally also a varying degree of parenchymatous and fatty degeneration of the muscle-fibres themselves. When the change is slight, complete recovery is possible, but otherwise, if the patient lives, these non-purulent infiltrations will result in fibrous bands and areas among the muscle-fibres, in other words, in a *fibrous* or *chronic interstitial myocarditis*, which has been already considered under the heading of *fibroid degeneration*.

Treatment.—When in acute endocarditis or in the course of diphtheria or similar infectious disease the myocardium is acutely involved, absolute rest in bed is necessary, and must be rigorously insisted on. Naturally the use of cardiac tonics and diffusible stimulants must be limited to the minimum quantity necessary, while poultices locally are of great value if pain is present. Where we can antagonise the poison, as in diphtheria, the antitoxin should be administered

so as to arrest, if possible, the advance of the condition. Strengthening soups, &c., must be frequently given in small quantities, and only when cardiac failure is imminent should we resort to general and cardiac stimulants.

In suppurative cases little can be done except to treat symptoms.

TUBERCULAR MYOCARDITIS.

Tubercular myocarditis is mainly chronic, being most often seen as a circumscribed caseous nodule of small or large (hen's egg) size, and much more rarely as a diffuse interstitial myocarditis whose true character is detected on microscopic examination by the tubercle foci (giant cells, &c.) seen here and there among the fibrous tissue. A more acute form—the miliary—in which the characteristic grey granulations are scattered through the heart muscle, also occurs. In nearly all cases there is tubercle elsewhere.

SYPHILITIC MYOCARDITIS.

This is also a chronic disease. It occurs most frequently as a more or less diffuse fibroid change, but also as gummatous deposits of greyish or yellowish colour of small or large (marble) size. The wall of the left ventricle is their most frequent seat, and they may cause an aneurism of the heart. Sclerosis of the coronary arteries and its resultant changes are also seen.

LESIONS OF THE CORONARY ARTERIES.

1. SCLEROSIS.—Thickening of the walls and narrowing of the lumen of the coronary arteries is of frequent occurrence. It may affect mainly their orifices or the larger or smaller branches. It diminishes slowly but progressively (except when it causes rapid thrombosis) the blood supply to the cardiac muscle, and consequently leads to fatty or fibroid changes therein.

2. EMBOLISM AND THROMBOSIS.—Thrombosis in the coronary arteries is more common than embolism, on account of the comparative frequency of atheromatous changes therein. The results vary with the degree and rapidity of production of the obstruction to the blood flow, and also with the size of the arterial branch affected. They may be grouped under two heads, according as the occlusion is quickly or slowly produced—(1) Sudden occlusion of a large branch, whether by embolism or thrombosis, may occasion instant death from cardiac failure, but if not they cause a patch of anæmic necrosis in the part supplied by the affected vessel, most commonly in the wall of the left ventricle, near the apex. When large enough it causes a slight elevation on the epicardial surface, and may be covered with soft thrombi on its endocardial surface; when small it may be deeply seated within the substance of the ventricular wall and escape notice unless carefully looked for. It is of a yellowish colour and firm consistence, and sometimes, when of longer duration, surrounded by a zone of redness, indicating inflammatory granulation tissue at its periphery. The patch itself shows necrosed muscle—in short, the changes seen in coagulative necrosis, referred to here as *Myomalacia Cordis*. It may form the seat of an acute aneurism and may end in rupture. In the more chronic cases the granulation tissue first formed at the periphery invades the whole

patch and transforms it into fibrous tissue. This may become the seat of a chronic aneurism. (2) Slowly produced occlusion of the coronary arteries, whether at the orifice or in the course of the main trunks, leads to fatty degeneration of the cardiac muscle, or to an interstitial myocarditis, or both. In most, if not in all, cases both processes are at work, the fibrous tissues thriving on the supply of nourishment, which gradually becomes insufficient for the more delicate and highly specialised muscle-cells. When the terminal twigs of the coronary arteries are affected the process is probably similar, though much more limited in extent.

HYPERTROPHY OF THE HEART.

DEFINITION.—Hypertrophy causes enlargement of the heart from an increase in its muscular substance, as distinguished from dilatation, which is so closely related to it, in which the enlargement is due to dilatation of its chambers. It may be *total*, involving all parts of the heart; *partial*, involving certain parts only; *simple*, when there is no accompanying dilatation; or *excentric*, when there is accompanying dilatation.

Etiology.—The thickness of the walls of the heart is in direct relation to the amount of work it requires to do in order to carry on the circulation satisfactorily. When any of the normal conditions of the circulation become altered so as to throw more strain upon the heart, it will, in healthy individuals, hypertrophy in order to do the increased work demanded of it. The hypertrophy will be total or partial, according as the cause acts upon one or all the chambers. Among the causes of total hypertrophy may be mentioned certain instances of valvular disease, long continued functional disturbance (such as tachycardia and palpitation), the over-use of tobacco, alcohol, tea, or coffee,

some cases of prolonged physical exertion, of chronic interstitial nephritis, and of adherent pericardium. Tobacco and alcohol may act partly by causing palpitation, partly by dilating the coronary arteries and thus increasing the nutrition of the heart. Chronic interstitial nephritis probably acts through first causing a thickening of the walls of the arterioles and capillaries, due to the altered condition of the blood brought about by the deficient renal excretion, and thereby throwing more strain upon the heart. This tells chiefly upon the left ventricle, and hence the hypertrophy is usually most marked in it. In the same way most, if not all, of the other causes mentioned above may mainly affect the *left ventricle*. Among the other or special causes of hypertrophy of the left ventricle are stenosis and incompetence (particularly the former) of the aortic valves, stenosis of the aorta, general arteriosclerosis (particularly when affecting the thoracic aorta or the splanchnic vessels). The special causes of hypertrophy of the right ventricle are any obstruction in the pulmonary circulation, either in the lungs, such as is produced in emphysema, fibroid changes in the lung, &c., or at the mitral valve, and stenosis of the pulmonary orifice or arterial trunk (rare and usually congenital). The left auricle may be hypertrophied in mitral stenosis and the right in tricuspid stenosis (rare) or obstruction to the pulmonary circulation. Accompanying dilatation is even more common in the auricles than in the ventricles.

Morbid Anatomy.—The weight of the heart (normally 9 to 10 oz. in woman and about 11 oz. in man) is increased, the thickness of the walls of the affected chambers is also greater, and the shape of the heart is altered. When the left ventricle is mainly affected the heart is elongated and the apex displaced to the left; when the right is chiefly affected the heart is broader and tends to become globular rather than cone-shaped.

Symptoms.—There are generally no symptoms so long as the hypertrophy is able to overcome and compensate for the increased resistance, but when it fails to do this there may be symptoms referable to imperfect blood supply to the brain or lungs, such as headache, noises in the ears, flashes of light, breathlessness on exertion, or cough, or palpitation. Examination of the chest shows an increased area of cardiac dulness in all cases, with, in addition, when the left ventricle is affected, the apex beat displaced downwards and to the left. When the right ventricle is greatly affected the apex beat may be formed entirely by the right ventricle. In the former the first sound is long and muffled and the second loud, clear and often reduplicated, in the latter the second sound in the pulmonary area is accentuated and often reduplicated.

Prognosis.—Hypertrophy is in itself a good thing, as it is Nature's response to an abnormal condition, and so long as it is able to compensate for and annul the consequences of that abnormal condition, the patient remains well, although his health equilibrium is of a less stable character.

DILATATION OF THE HEART.

DEFINITION.—An enlargement of the heart due to a dilatation of the cavities, with or without thickening of their walls. This definition includes cases of excentric hypertrophy where the dilatation is excessive. It may be total or partial.

Etiology.—It frequently accompanies or follows upon hypertrophy, when it is sometimes spoken of as *active dilatation*; but it may occur where there is no hypertrophy and even a thinning of the walls, sometimes then called *passive dilatation*. The causes of active dilatation are thus much the same as those of hypertrophy. In favourable cases hypertrophy results, in unfavourable cases (when the health

is low and the strain severe and sudden) dilatation, and in mixed cases a mixture of the two. When dilatation follows upon hypertrophy there may or may not be any structural change in the myocardium discernible by the microscope. In passive dilatation, on the other hand, such as occurs in the infectious fevers, &c., there are well-defined structural changes, *e.g.*, fatty and fibroid degenerations, which are the direct cause of the decreased muscular power, and hence of the dilatation.

Morbid Anatomy.—The size and shape more than the weight of the heart are affected. The size is increased and the shape will be altered according to the position of the dilatation. When total, the whole heart is increased, both in width and length, assuming a globular or quadrilateral shape; when the right or left ventricle are mainly affected the change in shape is much the same as in hypertrophy, only more marked. The mitral and tricuspid valves become incompetent from dilatation of the orifices, and the ventricles may be incompletely emptied at each systole.

Symptoms.—The symptoms are much the same as those of hypertrophy after the compensation fails. Pain, which is not usually present in hypertrophy, is often marked, particularly when sudden variations occur in the amount of dilatation. Dyspnoea, fainting fits, sleeplessness, dropsy and other signs of cardiac failure may supervene. The cardiac impulse is feebler and affects a wider area, and the cardiac dulness is generally greater in extent. The first sound may be short and sharp. Mitral and tricuspid diastolic murmurs appear when the corresponding orifices become dilated.

Treatment for Hypertrophy and Dilatation.—Much might be repeated here which is stated under the treatment of Valvular Lesions (page 458).

Cardiac hypertrophy is often associated with chronic renal and arterial disease, and the treatment consists in relieving the organ as far as possible from its excessive work by rest, careful dieting, and keeping up the free action of the emunctories, and especially the bowels and skin. The purely compensatory hypertrophy of valvular disease demands no treatment. Palpitation is, however, not infrequently present from excitement or overstrain, when aconite and other sedatives are often of use. Digitalis is contra-indicated, except when the palpitation is marked, suggesting the presence of commencing cardiac failure. Over-exertion should be avoided.

In dilatation, which is a confession of failure on the part of the heart muscle, the chief object is to improve the tone and vigour of the muscular walls of the heart, and also to support the strength of the patient. Digitalis may in this case, if cautiously given, be of great use. In the treatment of diseases of the heart no ordinary routine system should be adopted: each case must be treated on its merits. Oertel's and Schott's methods of treatment of the diseases of the heart by graduated exercises and baths are of very great value in some instances

The chief difficulty is to decide when rest in bed may be advantageously departed from and how much and what kind of exercise to allow in any given case. Experience alone teaches the physician when he is likely to overstep the bounds of prudence.

ANEURISM OF THE HEART.

DEFINITION—A local saccular dilatation of the cardiac walls.

Etiology.—It may arise in any part of the heart wall which has been weakened by disease. The most common causes are fibroid degeneration following upon coronary obstruction or a syphilitic gumma. The anterior wall

of the left ventricle near the apex is the commonest seat. The septum comes next in frequency, and aneurisms in this position bulge into the left ventricle. The aneurism varies in size, and may be very small or as large as an orange. Its mouth is usually wide, but occasionally it is very small, though the sac itself may be of some size. The wall of the sac varies with its age and position. Septal aneurisms usually show endocardium on each side, with myocardium between; while those bulging outwards are usually adherent to the pericardium, and though for a time they show the myocardium beneath, with an inner lining of endocardium, fibrous tissue changes appear in both layers and ultimately entirely replace them. The wall of the sac may rupture, or the aneurism may become stationary, and this is favoured by thrombosis in its interior. Even then the aneurism may contribute towards cardiac failure, which appears to be the commonest cause of death (much more so than rupture).

Symptoms.—If any symptoms are present, they generally suggest myocardial or endocardial trouble, and very rarely enable the presence of aneurism to be diagnosed during life.

Treatment.—The treatment must be purely palliative.

RUPTURE OF THE HEART.

Rupture may occur traumatically or spontaneously. The latter is very rare, and does not occur in a healthy heart. Fatty degeneration, due to stenosis of the coronary arteries, is the most common cardiac lesion which leads to rupture. Other causes have already been mentioned, such as aneurism, infarction, a syphilitic gumma, or a new growth in the wall of the heart. The rupture usually occurs during physical or mental excitement, and causes death instantly, or after

some time (several hours to several days), by interference with cardiac action, or possibly also by disturbed innervation, rarely by the actual loss of blood

TUMORS OF THE MYOCARDIUM.

Simple tumors—*e.g.*, myxoma, lipoma—are rare. Malignant tumors are more common. Cancers are always, and sarcomas nearly always, secondary: the latter appear most frequently as invasions of the cardiac walls from a primary growth in the mediastinum.

Symptoms.—There may be no symptoms when the growths are small, but large ones may cause cardiac dilatation, embolism, rupture, or valvular insufficiency, with their characteristic clinical results.

ANIMAL PARASITES.

The hydatid cyst is the only animal parasitic condition which becomes clinically important. It may be single or multiple, and the symptoms are much the same as those of tumors.

SUB GROUP (γ)—DISEASES OF ENDOCARDIUM.

DEGENERATIONS, notably fatty, are very common, particularly in the valves of the left side, but do not reach clinical importance.

INFLAMMATION.—Endocarditis is either an acute or chronic process. It is generally limited to a definite part of the endocardium, *viz.*, the valves, and not, as is usually

the case in pericarditis, of the general distribution, and the reason for this limitation is probably because the valves are the parts of the myocardium which bear the heaviest strain in cardiac action, hence the valves of the left side are much more frequently affected than those of the right in post-natal life, while the reverse is the case during foetal life. The proportion of left- to right-sided endocarditis in post-natal life is generally put too high, by some as high as 90 per cent. It is probably a good deal lower—about 60 per cent.—though in the greater number of the cases in which both are affected the left shows the greater degree, and hence from the clinical standpoint the older view is fairly correct. But this limitation to the valves is not the whole of the limitation shown by endocarditis, and probably for the same reason, viz., the distribution of the strain, for endocarditis is generally limited to that surface of the valves exposed to the direct current of the blood, viz., the auricular surface of the mitral and the ventricular surface of the aortic valves, and, further, it is not the whole of this surface but its line of maximum contact, and therefore of greatest strain (which is about $\frac{1}{16}$ inch above the free margin of the mitral valves and along the lower margin of the lunule in the aortic valves), which first shows the signs of endocarditis.

ACUTE ENDOCARDITIS.

It is still customary to divide acute endocarditis into two varieties, viz., simple and ulcerative, though it is now generally allowed that this division is based upon differences which do not exist. Micro-organisms were at one time believed to be absent in the former and present in the latter, whereas it has been demonstrated, both by experiments in animals and observations in the human subject, that

bacteria are present in both forms, and that acute endocarditis cannot arise without them. Notwithstanding this, however, the distinction into simple and ulcerative is still useful clinically, because the latter is the severer form and generally presents a fairly distinct clinical picture, and it can do no harm to continue to separate the two forms if it is remembered that they are but phases of the same pathological state, differing only in degree of severity, and that no hard and fast line can be drawn between them. Like inflammation in other situations, endocarditis may be set up by any one of the bacteria capable of causing inflammation, and the particular organism at work in each case may depend upon the primary disease or upon some secondary infection. Endocarditis is rarely, if ever, a primary lesion. It is an extension of a pathological process which begins elsewhere, as in the joints or in the lungs, caused either by the same organism spreading from the primary lesion to the endocardium, or by another inflammatory organism of later entrance, or by both. In the first class of cases the organism may belong to the cocci or the bacilli of general or non-specific inflammatory action, such as the pyogenic cocci or Fränkel's diplococcus, or to the specific group, such as the gonococcus or tubercle bacillus, and in the second class of cases it usually, if not always, belongs to the non-specific forms; while in some cases more than one kind of organism may be present at the same time or at different times in the same case. The number of organisms capable of setting up endocarditis is thus considerable; the staphylococci, the streptococci, the bacillus coli, the diplococcus lanceolatus of Fränkel, and the bacillus pyocyaneus among the non-specific forms, and the gonococcus, the diphtheria bacillus, the tubercle bacillus, the influenza bacillus, the anthrax bacillus and the bacillus typhosus among the specific forms. A number of observations have been made, chiefly upon fatal cases however,

with a view to determine which of these organisms is most often present, and these show that the diplococcus lanceolatus, the streptococcus and the staphylococcus pyogenes aureus are, in the order given, the most frequent. There is little or no difference between the organisms found in simple and malignant endocarditis.

It must not be inferred that bacteria are always found in the local lesions (the vegetations) of acute endocarditis, for some cases of the benign form have failed to show them; but their previous presence cannot be excluded, and pathologists are familiar with lesions of known bacterial causation which may fail to show the germs at the time of examination. For the reasons already given, it is now generally accepted that acute endocarditis is always of bacterial origin, but it may be otherwise with the chronic form. Toxins, whether of bacterial or of metabolic origin, existent for a long period in the circulating blood may cause degenerative or low inflammatory changes in the heart valves as elsewhere, which result in the condition known as chronic endocarditis, and it is also possible that such toxins may in many cases act mainly on the valves, or much more markedly upon them than elsewhere, owing to the perpetual strain thrown upon them. Attention must now be given to the diseases in which endocarditis is but an incident or a complication, and acute articular rheumatism is the most frequent of them, the approximate number of cases in which endocarditis has accompanied or followed it being about 40 to 50 per cent. It is more liable to appear after first and severe multiarticular acute rheumatism than after recurrent and mild attacks, but it may appear in cases so mild as to be overlooked, particularly in children, who are more prone than adults to develop cardiac lesions. Rheumatic endocarditis oftenest attacks the mitral valve, and rarely develops into the malignant variety. Chorea is the next most frequent disease, being little behind acute rheumatism, to which indeed, it is

suspected of being closely allied, and here also the endocarditis nearly always is benign. Other diseases, also believed to be related to rheumatism, in which acute endocarditis occasionally appears are tonsillitis, erythema nodosum and purpura or peliosis rheumatica. A number of other diseases may be grouped next in order, viz., septic and pyæmic conditions, scarlet fever, lobar pneumonia, measles or other specific fever. Other diseases have still to be mentioned, viz., gonorrhœa (in which the vegetations may be caused by the gonococcus or by an organism of secondary infection, such as a staphylococcus or a streptococcus, or by a combination of these), tuberculosis (especially in cases of acute general tuberculosis, but even in cases of phthisis), diphtheria, influenza, and after certain injuries to the chest and other regions from subsequent infection with some organisms. In all these diseases the form of the endocarditis may be either benign or malignant, but in gonorrhœa it is nearly always malignant, and in lobar pneumonia, septic infection and meningitis it is also frequently malignant. Lastly, there still remains to be mentioned wasting diseases generally, such as cancer, diabetes, chronic Bright's disease and alcoholism, leading to a lessened body resistance, in which benign endocarditis may appear from a late or terminal infection. For the purposes of convenience the morbid appearances, symptoms, &c, will be given under the headings of "Benign Endocarditis" and "Malignant Endocarditis."

BENIGN ENDOCARDITIS.

Syn. Simple, Warty, Vegetative.

Morbid Anatomy.—Small, slightly rough areas first appear along the lines of maximum contact of the valves, soon to become small, raised, grey, semi-transparent projections—a chain of minute vegetations, which are at

first soft and friable, but later become whiter and firmer. The vegetations are simply white thrombi, and they may remain small for a long time, or they may increase in size and join in places to form one or more large projections of an irregular nodular or polypoid appearance, with smaller ones in their neighbourhood. Occasionally the vegetations extend to the chordæ tendinæ or adjacent parts of the endocardium. The valves generally appear to be unchanged otherwise, but there is no doubt that there is always some change in their structure. The process probably starts in minute areas of necrosis which involve the endothelial cells and a varying amount of subjacent tissue. Fibrin is deposited on these areas from the circulating blood, not from the valves themselves, thus forming the nucleus of the vegetation, which gradually increases in size from the deposition of more fibrin and cells from the blood which flows over it. Around the necrotic focus a collection of cells presently appears, derived, in part at any rate, from proliferation of the connective tissue cells of the valves, and in part, it may be, from leucocytes. In the vegetations, and sometimes near these inflammatory cells, bacteria are generally to be found. So long as the acute stage continues, more of the valve may be affected and the vegetations may increase in size; but when it subsides a chronic stage may supervene, when these inflammatory cells will gradually replace, by newly-formed fibrous tissue, the necrosed part of the valve, and invading also the substance of the vegetation, convert it into fibrous tissue, leaving a permanently thickened or deformed valve. This is, however, a result and not a part of the acute process. Another result may be that, although absorption may go on, fresh deposit may occur, and the new fibrous tissue formation extensively invades the valve and projects considerably above the surface; or fatty or calcareous degeneration may occur in the thickened valve and the adherent clot.

Symptoms.—It is often difficult to distinguish the symptoms due to the endocarditis from those caused by the primary disease. It is to be remembered, further, that the active agent of the primary disease may act also upon the myocardium and cause an accompanying myocarditis, to which the cardiac symptoms present may be due; but the endocarditis *per se* may give rise to certain clinical results, ascribable either to the local cardiac lesion or to other and distant organs from something produced locally getting into the circulation. The local lesion may conceivably at times obstruct the onward flow of blood, but few believe that it ever does so to such an extent as to be detectable by any symptom or sign, or it may render the valves incompetent and allow of regurgitation of the blood into the chamber behind. The heart sounds may be muffled, or a systolic murmur may appear in the mitral area and a diastolic in the aorta, caused in most cases doubtless by functional incompetence of the valves through dilatation of the orifice. If palpitation and irregular heart action be present, they are probably due to a coincident myocarditis. As regards what the circulating blood may receive from the vegetations, there is, firstly, the toxins of the bacteria in the vegetations, and, secondly, bits of the vegetations themselves breaking off and becoming emboli. The former are rarely intense enough to cause appreciable trouble, but the latter is a common cause of embolism in the brain, kidneys, spleen, limbs, &c.

Diagnosis.—Valvular murmurs and embolism are the most valuable signs, *vide* the malignant form.

Prognosis.—Many cases steadily progress and lead to cardiac failure and death sooner or later, but others subside and become chronic, and some recover. Were it not for the constant movement recovery might occur much oftener than it does. It is more common upon

the right than upon the left side, for vegetations are to be found on the right valves in a large proportion of cases of acute endocarditis, probably about 40 per cent. of the whole, whereas, while left-sided chronic endocarditis is very common, right-sided chronic endocarditis is very rare.

Treatment.—The moment that we suspect endocarditis to be present, absolute rest in bed is necessary, and the areas of the heart should be auscultated frequently and carefully. In all diseases commonly associated with endocarditis, investigation of the heart sounds is an imperative duty. Many cases are of rheumatic origin, and salicylates and alkalies should be administered, whether we espouse the belief that salicylates have a specific action or not. The heart, if excited, should be calmed with such sedatives as the tincture of aconite (m 2 to 5), or, better, potassium iodide (10 grains). Pain may call for the use of opium or bromides; but both cardiac excitement and pain are well controlled with the icebag. Blisters locally have been recommended. Above all, rest must be enjoined until the inflammatory process has subsided, and if this has only been achieved with organic changes in the heart, compensation should be as perfect as possible for the new conditions before any freedom of movement is permitted (see treatment of "Valvular Diseases").

MALIGNANT ENDOCARDITIS.

Syn. Ulcerative, Septic, Suppurative, Infective.

Morbid Anatomy.—The general appearances are much the same as in the other form, but in typical cases certain differences may be noted. The vegetations may be larger, more irregular in shape, softer and more friable. They generally extend over a greater area of the valves and

show a greater tendency to spread to the septal or other adjacent endocardial surface. The substance of the valves is more implicated, showing more extensive necrosis, leading often to acute aneurism, ulceration or perforation of the valves or rupture of chordæ tendinæ; in short, the destructive process is in excess of the reparative, whereas the reverse is usually the case in the benignant form. The accompanying myocarditis is greater and may be suppurative. The number of bacteria present, both in the vegetations and in the affected parts of the valves, is usually greater. Certain cases of malignant endocarditis occur where no preceding disease or lesion can be discovered, and those cases may be regarded as themselves primary, or as of cryptogenetic origin. Sclerosed valves are more prone to be attacked than healthy ones.

Symptoms.—The symptoms due to the accompanying myocarditis (*vide* page 422) are more pronounced than in the benignant form, and those caused by the endocarditis are also more severe. The local valvular lesion may cause murmurs which change their character from time to time; but the important difference in the symptoms lies in the severity of the results of toxin absorption and the greater frequency and seriousness of embolism. The former, always present, though varying greatly in character and intensity, resemble those of septicæmia or pyæmia (*vide* page 137), and the latter, often absent, those of embolism of the different organs implicated, such as sudden pain and enlargement when the spleen or kidney is affected (accompanied in the latter case by albuminuria, and often hæmaturia), or apoplexy, sudden paralysis, &c., when the brain is involved. The acuteness of the symptoms and the course of malignant endocarditis vary greatly. In the cases where it attacks sclerosed valves, which form, indeed, the majority (the so-called cardiac cases, because of the prominence of cardiac signs), it often runs a

somewhat prolonged course, even many months; but in a number even of these cardiac cases, and in most of the others, it generally proves fatal within five or six weeks, and sometimes within a few days. It is believed that some at any rate of the cardiac cases may recover. Two types are recognised, viz., the septic or pyæmic and the typhoid, according as the chief symptoms resemble those either of septicæmia or of pyæmia on the one hand, or of typhoid fever on the other; but some other cases, in which the chief symptoms resemble those of meningitis, may be held to constitute a cerebral type, while the cardiac cases above referred to are similarly constituted into a cardiac type. In all diseases where toxin, germ or embolic invasion of the blood occurs, as in malignant endocarditis, one or other part of the body may be mainly affected, and the results of this particular affection may for a time overshadow all others, even those of the local cardiac lesion itself, which may be unsuspected or undetectable if suspected. In the typhoid type, which has already been stated to be the most common, the main incidence of the infection appears to fall upon the intestine, the skin and the brain, causing diarrhœa, profuse sweating and small hæmorrhages in the skin, continued and irregular fever, early prostration, somnolence, delirium or coma towards the end—a picture very like that of typhoid fever. In the next most frequent type—the septic or pyæmic—there is a more general incidence of infection, and though the same symptoms may be observed, others may appear, such as more frequent rigors, particularly where metastatic abscesses are formed or arthritis occurs, more irregular fever, a more frequent occurrence of a recognisable source of infection (a septic wound, acute osteomyelitis, the puerperium, &c.); in short, a course like that of septicæmia or pyæmia, of which malignant endocarditis is merely a particular form.

Diagnosis.—It is often extremely difficult and cannot be made in the absence of signs of valvulitis. Palpitation, cardiac pain, tendency to fainting, breathlessness are more likely to be due to myocarditis than endocarditis, and hence cardiac murmurs are chiefly to be relied on. The varying character of the murmurs may be of assistance, and the occurrence of embolism, combined with the gravity of the general symptoms, is of great value. It is most likely to be mistaken for typhoid fever, from which it may be distinguished by the Widal test or by a bacteriological examination of the blood or urine for the specific bacillus of typhoid fever or other organism. It must not be forgotten that malignant endocarditis may, though it very rarely does, appear in the course of an attack of typhoid fever. Other cases resemble typhus, small-pox, cerebro-spinal fever, and a differential diagnosis may be possible only after patient observation.

Prognosis.—The disease is almost always fatal. Two days is the shortest duration recorded, but a few days' duration is not uncommon, and cases rarely live longer than five or six weeks, though some last for months, and a few of these may recover.

Treatment.—While much might be repeated which has been already referred to under the treatment of the benign variety of endocarditis, there are a few special points to be mentioned. Treat the case as one of septicæmia or pyæmia, enforce the most absolute rest, and if a streptococcus be present in the blood try antistreptococcic serum, beginning with 5 c.c. and rapidly increasing till 15 to 20 c.c. are given daily. In several cases we have seen much benefit derived from the serum, and certainly the heart has been saved from greater damage if we cannot assert that by it alone a fatal result was averted.

CHRONIC ENDOCARDITIS.

Etiology.—1. Acute endocarditis, especially of rheumatic origin, is its most common cause. An interval generally intervenes. Usually the acute disease subsides and the chronic form appears slowly, gradually and insidiously, the first signs appearing perhaps years afterwards. When the acute disease has been so mild as to have escaped notice altogether, as it may well do, not only in rheumatism, but also in scarlet fever, measles and others of the infections mentioned on page 435, the subsequent chronic disease may be falsely regarded as primary. Care must be taken not to date the commencement of the disease from the date of the first symptoms. It may have been in existence for months and even years before that.

2. This raises the question of the possibility of chronic endocarditis arising in the course of these infectious diseases without acute endocarditis preceding it. It is even reasonable to suggest that certain poisons, if circulating long enough in the blood, and diffusing into the lymph-stream of the valves, may cause degenerative or low inflammatory changes of a much more pronounced character in the valves than anywhere else, because of the constant movement and strain to which they are subjected. Between degenerative and low inflammatory changes no hard and fast line can be drawn, and both may cause sclerosis of the valves. The chief objection to such changes arising during the acute infections is the comparatively short duration of the presence of their specific poisons within the blood. It is not, however, impossible for a poison to start degenerative changes which may gradually progress and continue long after the originating poison has disappeared. Acute infections may, moreover, start altered metabolism, which may persist and affect nutrition long after the acute infections themselves have disappeared.

3. Gout, alcoholism and syphilis are other examples of diseases which are believed to be causes of chronic endocarditis, and they do so probably by their poisons acting slowly upon the valves, as already suggested.

4. Strain is another cause, particularly in the aortic valves, where it is brought on by overuse of the muscles, as in soldiers, labourers, athletes, &c. It probably acts by impairing the nutrition and thus causing degeneration in places, the products of such degenerations stimulating the surrounding tissues to over-production.

Morbid Anatomy.—In the slightest cases there are nodular or diffuse thickenings in the valves somewhere along the line of maximum contact. They may be hardly palpable but they are always easily recognisable by their increased opacity. They may, on the other hand, form very obvious thickenings, and the greater degrees extend over a greater area of the valve surfaces. The nodules are always firm whitish or whitish-yellow, often hard and cartilaginous. Calcareous salts may be deposited in them. The thickening soon involves the free margin of the valves and causes them to curl, and makes their accurate apposition impossible. This leads to incompetence of the valve, *i.e.*, imperfect closure of the orifice, *vide* below, but not necessarily to any stenosis or narrowing of the orifice. At the aortic orifice incompetence without stenosis is common, at the mitral orifice it is much less so. In this latter position the thickening generally involves the contiguous edges of the two valves at their angles, causing adhesion between them, which may be present in any degree, either slight or complete, and thus a corresponding degree of stenosis. When the adhesion between the two mitral cusps is complete, it gives rise to a funnel-shaped process projecting into the ventricle, at whose apex is the mitral orifice. This is common in children, and is sometimes called the funnel-shaped mitral. It occurs also in adults, but it is usually

accompanied by a greater thickening and contraction of the valves, and hence of the mitral orifice, which may be reduced to a mere slit—the button-hole mitral. The chordæ tendinæ are likewise thickened and shortened, and in extreme cases the apices of the papillary muscles are themselves fibroid. In the aortic orifice, on the other hand, fusion between the valve cusps may exist without stenosis, though it often causes it here also. An acute endocarditis may at any time appear upon the chronic lesions. In its early stages a chronic endocarditis in most cases leads to an imperfect closure of the orifice which the valves are meant to guard. The thickening prevents the perfect elasticity and apposition necessary to completely close the orifice, and thus some of the blood is allowed to flow through it into the chamber behind, *e.g.*, from the left ventricle into the left auricle during ventricular systole, instead of its all going through the aortic orifice into the aorta. When this regurgitation occurs, the valves are said to be incompetent, and the left auricle has to find room for this regurgitated blood plus its ordinary supply from the pulmonary veins. The amount regurgitated during each ventricular systole must be very small at first in most, if not in all, cases, but it will tend to increase, and in time it may become considerable. To accommodate this increased quantity of blood the auricle must dilate. The dilatation of its cavity is usually accompanied by a certain degree of hypertrophy of its wall in order to be able to pump the increased quantity of blood into the ventricle. In most cases this dilatation and hypertrophy proceed step by step together. The left auricle is greatly aided in this by the right ventricle (which is indeed the more important factor). The blood regurgitating through the mitral orifice impedes the free outflow of blood from the pulmonary veins, and the circulation of blood through the lungs would necessarily become slower if the right ventricle did not answer to the strain, and by hypertrophying enable the blood to be driven

through the lungs and the mitral orifice, aided by the left auricle, within the normal time.

The results of the lesion are annulled for the time being, the hypertrophy having *compensated* or made amends for it. This *compensation* is a most important factor in all chronic valvular lesions. It is always to be hoped for, and so long as it holds good the patient is as sound as he was before the lesion began, but the stability of his equilibrium is not so good. The compensation is liable to be upset from time to time, causing various temporary symptoms, and ultimately it is likely to be permanently disturbed, when evidence of cardiac failure in some form appears and persists. The study of chronic endocarditis, as it affects the various valves, is practically the study of the various chronic valvular lesions. These lesions lead to either or both of two circulatory disturbances, viz., stenosis and incompetence, at any of the four cardiac orifices, which, on account of their frequency and importance, require detailed notice. Aortic and mitral incompetence and stenosis are the four most common cardiac lesions met with in practice, and of these the two incompetences are far more important because more frequent than the two forms of stenosis. Some authorities say that aortic, others that mitral incompetence is the most frequent of all the cardiac lesions.

MITRAL INCOMPETENCE.

Etiology.—The remarks already made upon the etiology of chronic endocarditis apply to all the valves; but some of these causes show a selective affinity for certain valves, the mitral, for instance, suffering after rheumatic fever far more frequently than the aortic.

Morbid Anatomy.—The valves are either thickened, contracted or distorted as the result of endocarditis, or they

are normal but their orifice dilated. In the first instance, sometimes called organic incompetence, the mitral orifice may be of normal size, for a time at any rate, but the valves having become smaller and incapable of perfect apposition can no longer completely close the orifice. In cases of more acute production there may even be dilatation of the orifice, thus accentuating the incompetence; but in chronic cases the contraction of the valves generally (some say always) involves also the orifice, thus lessening or even avoiding incompetence for a time. In the second instance, called functional or relative incompetence, either the mitral orifice (which is guarded by a ring of tissue, mainly muscular) is dilated so that it can no longer be completely closed by the valves, which themselves remain healthy, or the cardiac muscles contract irregularly and prevent the perfect apposition of the valves. This dilatation of the orifice or imperfect cardiac contraction may arise in any disease which leads to lessened nutrition, and thus to diminished tonicity and strength in the cardiac muscles, such as all anæmias, whether primary or secondary, and all general infectious diseases. Dilatation and hypertrophy of the left auricle results from the incompetence. Congestion of the lungs follows, and will continue until hypertrophy of the right ventricle becomes strong enough to overcome it. When it does so, compensation is complete. This is usually the case during the ordinary development and course of the lesion. It may last for years or be disturbed temporarily or fail permanently. The left ventricle usually becomes dilated, owing to its receiving more than the normal amount of blood at each auricular systole, and hypertrophied, owing to its having to pump that increased amount into the aorta. The right auricle is unaffected. When compensation is temporarily disturbed, either through unwonted muscular effort or through some weakening illness, congestion of the lungs (which may cause breathlessness, cough, spitting of blood, &c.) and, later, of the

larger systemic circulation may follow. The latter is due to dilatation of the right ventricle causing incompetence of the tricuspid valves and consequent dilatation and congestion of the right auricle. When compensation permanently fails, all these results become more pronounced and lasting. The systemic venous congestion shows itself in congestion of the liver (enlargement and tenderness), spleen, kidneys (albuminuria and hæmaturia), and limbs (dropsy), &c.

Symptoms.—(1) During perfect compensation there are no subjective symptoms, or if any do appear at intervals they are due to temporary embarrassments of the pulmonary circulation, and are directly referable to some unusual exertion or a respiratory catarrh. Shortness of breath on exertion is the most frequent symptom, and there may be some cyanosis of the face, and in children clubbing of the fingers. The more pressing symptoms soon disappear under rest and proper treatment. The physical signs, on the other hand, are pronounced. Inspection may show a widely-spread cardiac impulse. Palpation usually discovers the apex beat to be displaced downwards and outwards, the degree of displacement depending upon the degree of dilatation of the left ventricle. The cardiac impulse may be forcible, and thus a systolic thrill may be felt at the apex. Percussion gives an increased area of cardiac dulness. Auscultation demonstrates the presence of a mitral systolic murmur, either blowing, rough or musical, generally heard best in the mitral area and propagated for a certain distance all round, particularly into the left axilla and the angle of the left scapula (posterior aspect of left auricle). Sometimes it is heard only when the patient is lying on his back, and at other times it is heard over the whole back or at the angle of the right scapular, and very rarely disappears permanently. There is also accentuation of the second sound in the pulmonary area. The pulse is usually full and regular. (2) When compensation fails, symptoms

become more or less marked. Breathlessness and dyspnœa appear. The patient is apt to wake with a start, just as he is falling off to sleep, with a feeling of sinking or as if his heart were stopping. There may be palpitation and irregular heart beat. There may be cough with a copious watery or hæmorrhagic expectoration. Dropsy of the feet sets in, gradually spreading up the legs to the body and serous cavities. Hepatic, splenic, gastric, intestinal and renal congestion occur. The physical signs also change, the cardiac impulse becoming feeble and wavy, the dulness usually increasing in area and the murmur becoming lost, or heard only at intervals. The pulse becomes irregular and weak.

Diagnosis.—The signs and symptoms above mentioned, particularly the murmur, the accentuated second sound, and the enlargement of the heart, suffice in most cases to make the diagnosis clear. The history of the case is of the greatest value in determining whether the mitral insufficiency is due to relative or organic incompetency of the valve.

Prognosis.—Efficient compensation is the keynote of prognosis in all cases of valvular lesions. There is no trouble so long as this is maintained, and temporary disturbances thereof, with their results, frequently disappear under appropriate treatment. Some cases, especially in children, go more or less rapidly downhill, but others, when the circumstances are favourable, live for years, even thirty to forty years, and may reach old age without serious inconvenience. Death is rarely sudden; it is generally due to advancing dropsy. Speaking generally, the outlook is considerably better than in aortic insufficiency, better even than in mitral stenosis.

MITRAL STENOSIS.

Etiology.—Acute rheumatism or chorea is answerable for the majority of cases of mitral stenosis, and this may account for the greater frequency of the disease in girls than boys, and in early adult life, say fifteen to twenty-five. But there are other cases in which no preceding disease likely to have a causal relation to the valvular stenosis can be traced, and in them the etiology is obscure, although it is to be remembered that many of them may be explained by a slow and unsuspected development from some acute infectious mischief much earlier in life.

Morbid Anatomy.—Thickening and deformity of the mitral valves and contraction of the auriculo-ventricular orifice, with adhesions of the cusps, as already described, are the lesions found in these cases. The left auricle is hypertrophied and dilated. Many authorities describe white thrombi in the appendix, and even occupying a large part of the auricle, as not uncommon. Others have met with them very rarely. The lungs are congested whenever the consequent hypertrophy of the right ventricle fails, as it may do under any unusual strain, to overcome the obstruction at the mitral orifice. The left ventricle is of normal size, and may even be slightly smaller than normal in some cases. When compensation completely fails, the right ventricle becomes dilated out of proportion to its hypertrophy. Dilatation of the right auricle, general venous engorgement, and dropsy follow, as in mitral incompetence. Occasionally an acute endocarditis, which is at all times liable to arise upon sclerosed valves, follows, and it may cause embolism and infarction with their characteristic phenomena.

Symptoms.—There are no subjective symptoms when compensation is established, as it usually is. There may be some shortness of breath during unusual exercise, such as running up stairs, and there may be cough and even severe hæmoptysis at times from bronchial and respiratory catarrh or causes which disturb temporarily the compensatory action of the right ventricle; but at other times the patient is well and may reach old age. After compensation fails permanently, the same train of symptoms are seen as in mitral incompetence.

PHYSICAL SIGNS.—*During compensation.* *Inspection:* As a rule nothing unusual is seen, but there may be pulsation in the third and fourth left interspaces close to the sternum. *Palpation:* A presystolic thrill is usually present in the fourth and fifth interspaces within the nipple line. It is pathognomonic. It is best felt during expiration, and may last during the greater part of the diastolic period. The cardiac impulse follows it closely, and is short and sudden, and felt best in the same interspaces. *Percussion:* There is generally some increase in the area of dulness to the right. *Auscultation:* A rough murmur, presystolic in time, is heard best in the mitral area, to which it is practically limited. It runs into and terminates abruptly in the first sound, which is sudden, sharp and clear. The second sound in the second left interspace is accentuated and often reduplicated. There may also be a mitral diastolic murmur. These murmurs and thrill may disappear in the recumbent position, but may be made to reappear after standing up or after exercise, such as running up stairs. The pulse is small and usually regular. When compensation fails, the thrill and murmur usually disappear, the first sound is less sharp and clear, and cardiac action becomes irregular. A systolic murmur may appear in the tricuspid area after dilatation of the right ventricle.

AORTIC INCOMPETENCE.

Etiology.—It is much more directly related to strain than are lesions of the mitral valve, and hence it is most commonly met with in males during middle and advanced life, especially those who follow laborious occupations. Chronic endocarditis, mostly acquired and rarely congenital, is the most frequent condition present in the valves; but its antecedent excitant is much more commonly strain, alcohol, or syphilis than rheumatism. In some cases it is rather a chronic endarteritis of the aortic arch, which spreads to the valves, setting up changes therein closely resembling those of chronic endocarditis, and the changes often exist together. Acute endocarditis does not in its acute stages cause incompetence unless there is perforation, rupture, or extensive destruction of one or other of the valve segments. The incompetence in all these cases is due to the alteration in the valves. The occurrence of a relative incompetence from a simple dilatation of the aortic ring, consequent upon relaxation due to dilatation of the left ventricle, is of rare occurrence, if it exists at all, and is thus in marked contrast to the mitral orifice where it is common.

Morbid Anatomy.—The morbid changes in the valves are those already described under acute and chronic endocarditis respectively. When there is a concomitant endarteritis, there is often considerable dilatation of the aortic arch and some stenosis of the coronary arteries. The regurgitation causes dilatation of the left ventricle, which follows all the more readily if stenosis of its coronary arteries is present as well. Hypertrophy follows the dilatation in most cases, particularly when the nutrition of the heart is good. Compensation is established by the preservation of the balance between

dilatation and hypertrophy, and may remain good for years. The enlargement of the left ventricle may be very great, and may go on increasing for a long time without failure of compensation. Temporary disturbances may occur, which lead to dilatation and hypertrophy of the left auricle, and later of the right ventricle, which in turn restore compensation. Very large hearts (35 to 40 oz.) may thus be produced. When compensation fails, there is further dilatation of the left ventricle and of the other chambers, and all the evil effects of backward pressure as already described. When the mitral valves are also sclerosed, the establishment of compensation is more difficult, and it does not usually last so long.

Symptoms.—During compensation there are usually no symptoms. There may be occasional giddiness, flashes of light, and even syncope, due to anæmia of the brain, or cardiac pain and distress, due to dilatation of the left ventricle and aorta, or perhaps to changes in the cardiac nerves or ganglia.

PHYSICAL SIGNS.—*Inspection and palpation:* The area of cardiac impulse is larger. The apex beat is displaced downwards and outwards. A diastolic thrill may be felt, but it is rare. *Percussion* shows the cardiac dulness to be increased, especially downwards and to the left. *Auscultation:* A diastolic murmur is heard at the junction of the second right intercostal cartilage with the sternum, or in the second right interspace, which is propagated upwards towards the neck and downwards along the left margin of the sternum and towards the apex. The murmur is soft and blowing, and usually replaces the second sound, though in some cases both are heard. While it is the most characteristic and reliable murmur, there may be others also present, viz., a systolic aortic, a presystolic mitral, and a systolic mitral. The first is the most frequent. It is short and rough, and is

believed to be due to roughening of the aortic cusps or of the intima of the aortic arch, and not to stenosis of the aortic orifice. The second, called Flint's murmur, is roughish, but not so much so nor so distinct nor so regular as to rhythm as the presystolic murmur of mitral stenosis, though, like it, it is heard over a limited area. It is supposed to be due to interference by the regurgitating aortic blood with the anterior mitral cusp during ventricular diastole, and thus during flow from auricle into ventricle. The third murmur is the ordinary systolic mitral murmur, due to dilatation of the mitral orifice. When compensation fails, the usual symptoms of cerebral anæmia and pulmonary and systemic venous congestion gradually supervene. Pain may be a marked feature, and true angina is commoner than in any other cardiac lesion. Dyspnœa may be very troublesome, particularly at night. It may be relieved by the patient sleeping in the sitting posture. Sudden death is not uncommon, and is due to syncope or angina. Rupture of a diseased valve by sudden strain may prove fatal. The pulse gives most important evidence in this disease. The rise is sudden and jerking, and it collapses immediately—the so-called Corrigan or water-hammer pulse; this is best appreciated by bending the arm upwards. Pulsation may be observable in all the arteries, particularly in the carotids. Capillary pulsation is usually easily demonstrated in the forehead or in the finger-nails. If a line be drawn firmly across the forehead the margin of hyperæmia on either side of it alternately flushes and pales.

Diagnosis.—The enlargement of the heart, the aortic diastolic murmur, and the radial and capillary pulses usually serve to make the condition clear.

Prognosis.—The duration of life is on the average usually shorter than that of other forms of valvular disease,

but many cases last for years with good general health even under the strain of a fairly busy life. The co-existence of mitral disease or of general arterial sclerosis or of angina pectoris tends to hasten cardiac failure.

AORTIC STENOSIS.

Etiology.—The causes are much the same as in aortic incompetence, although it seems to appear generally later in life and to be accompanied more frequently by extensive arterial degeneration.

Morbid Anatomy.—The valves are most frequently thickened, sclerosed, adherent and rigid. The extent of the change is sometimes very great and may show itself as a nodular or diffuse cartilaginous or calcareous sclerosis reducing the aperture to small dimensions. In other cases there is adhesion without much thickening of the cusps, thought to be chiefly the result of foetal endocarditis. A pure stenosis is generally believed to be rare, there being nearly, if not always, a certain amount of incompetence as well. When the first part of the aortic arch immediately above the valves is greatly dilated (as the result of endarteritis) and the aortic ring and its valves remain normal, it is sometimes spoken of as a *relative* aortic stenosis. The left ventricle shows a hypertrophy varying in degree according to circumstances. It is greatest when the aortic lesion is very gradually produced and the nutrition of the ventricle is good. If in such cases there be no or very slight incompetence, there is little or no accompanying dilatation. The hypertrophy is the main thing and the consequent compensation is good. When incompetence is also present

there is always dilatation as well as hypertrophy, but this does not interfere with the establishment of compensation in favourable cases. With failure of compensation the same morbid changes follow as in aortic incompetence.

Symptoms.—During compensation there are usually no symptoms except after some unusual demand is made upon the heart by muscular effort, when there may be faintness, giddiness, palpitation, or pain.

PHYSICAL SIGNS.—*Inspection*: There may be nothing abnormal to be seen, or there may be a very distinct cardiac impulse and displaced apex beat. *Palpation*: There is often a well-marked thrill in the aortic area, and the apex beat may be felt to be displaced downwards and outwards; but it is often difficult to detect it, even when the left ventricle is dilated, owing to the emphysema which may be present. *Percussion*: The area of cardiac dulness may show no, or a considerable, increase, according to the amount of dilatation of the ventricle; but it is often masked by lung emphysema. *Auscultation*: The typical murmur of this lesion is a systolic one, heard best in the aortic area and propagated into the great vessels at the root of the neck. It is always to be remembered, however, that an aortic systolic murmur is produced much more often by other lesions than by a narrowing of the aortic orifice, notably by roughening of the valves or of the aortic arch, and by anaemia. Other murmurs may also be present, e.g., a diastolic aortic and a diastolic mitral. Neither of the heart sounds may be heard at the aortic area, but if there be dilatation and roughening of the aorta, the second aortic sound may be accentuated and ringing in quality. The pulse is usually small, slow, regular and of good tension. When compensation fails, the symptoms and physical signs become similar to those of failure in aortic incompetence. Sudden death occurs sometimes.

Diagnosis.—The rough systolic aortic murmur, the thrill, and the slow pulse of moderate volume are the most prominent guides; but attention must be paid to all the facts of the case, as mistakes are common.

Prognosis.—Many cases do very well and do not appear to limit the duration of life. Extensive arterial degeneration makes the outlook much graver in this than in other cardiac lesions.

TRICUSPID REGURGITATION.

Etiology.—In by far the majority of cases it is relative and due to the dilatation of the right ventricle, which in turn is mostly caused by chronic pulmonary or by mitral valve disease. In the small minority of cases it is due to changes in the valves, caused either by acute or chronic endocarditis.

Morbid Anatomy.—There is dilatation of the orifice without change in the valves in most cases, as already mentioned; in the few there are thickened and sclerosed valves. The right auricle and ventricle are both dilated and hypertrophied

Symptoms and Signs.—There is a tricuspid systolic murmur, and generally marked pulsation in the jugular veins, and gradually all the signs of cardiac failure appear.

TRICUSPID STENOSIS.

Etiology.—It may be either congenital or acquired. In the great majority of the cases it appears to follow upon lesions of the mitral valve.

Symptoms and Signs.—A presystolic murmur, heard best at the base of the ensiform cartilage, is the most characteristic sign, but it may be absent. There may be cyanosis of the face and lips, and general systemic venous congestion and dropsy may be very great. In most cases its symptoms complicate and intensify those of mitral stenosis.

PULMONARY STENOSIS (*rare*).

Etiology.—It is almost always the result of foetal endocarditis, but it may occasionally be of rheumatic or septic origin.

Morbid Anatomy.—The valves are sclerosed and adherent, leaving a narrow orifice. In the congenital cases there may also be a patent ductus arteriosus, an imperfect interventricular septum, and a patent foramen ovale. The right ventricle is hypertrophied

Symptoms and Signs.—A systolic pulmonary murmur, not transmitted upwards, and thrill may be present. There may be dyspnoea and cardiac distress and pain.

PULMONARY INCOMPETENCE (*rare*).

Etiology.—It is also mostly congenital, but it may be septic (malignant endocarditis) or traumatic in origin. It may be relative from dilatation of the pulmonary orifice, the valves remaining normal.

Symptoms and Signs.—A pulmonary diastolic murmur is the characteristic sign, but it is difficult to distinguish it from an aortic diastolic. When the murmur is soft and

there is high pressure in the pulmonary artery there may be temporary leakage or regurgitation through the valves, from stretching of the orifice (relative or functional incompetence).

COMBINED VALVULAR LESIONS.

Two different kinds of combined lesions have to be considered—(1) Those which follow upon the primary valvular lesion as a result of the dilatation of one or more of the cardiac chambers; (2) those that are produced more or less together by the same exciting cause. Almost every cardiac case shows at some period or periods of its course combinations belonging to the first group, and others of the second group are by no means uncommon, *e.g.*, combined affections of the aortic and mitral, of the tricuspid and mitral. Their results and symptoms are generally combinations of those of the separate lesions, and correspondingly more serious.

Treatment of Valvular Lesions.—The treatment of valvular disease falls naturally under two subdivisions—(1) If compensation is perfect, no direct treatment by drugs is requisite; but, on the other hand, the physician may be called upon to define the rules to be followed as regards work, exercise, and possibly diet, and certainly should enjoin keeping in free action the emunctories, so as to prevent a breakdown. The more serious the lesion the more rigid and careful should such rules be, even where compensation is perfectly adequate, because the greater the valvular difficulties the less will be the residual energy of the heart, and the more likely will any trifling overstrain bring about failure of compensation. (2) If compensation has failed, treatment must be directed to its immediate recovery with the least possible hypertrophy of the dilated chambers

and a minimum of change from the normal condition of the heart. We may find symptoms indicating (*a*) increased backward pressure, as in mitral lesions, or (*b*) diminished forward pressure, as in aortic incompetence, and we must, in addition, treat symptoms which may be present, such as cardiac pain. It should be remembered that with dilatation of one or more chambers of the heart, degenerative changes are apt to occur in the myocardium, and also that much which must be said here as to the treatment of cardiac inadequacy will equally apply to cases where the primary lesion is a myocardial and not a valvular one.

When compensation has broken down, invariably try first absolute rest in bed, and often for a prolonged period. Very great dilatation may disappear almost entirely by this means alone. Next relieve the backward pressure, if present, by giving diuretics (diuretin 10 grain doses, the spirit of nitrous ether in 20 to 30 minim doses, and other remedies), hydragogue cathartics (magnesium sulphate in 1 to 4 drachm doses, compound jalap powder in 20 to 40 grain doses), and more rarely by diaphoretics, and if these fail or are insufficient, either remove œdematous fluid from pleura, peritoneum or subcutaneous tissues or perform venesection. Twenty to thirty ounces of blood drawn from the arm may not merely relieve the patient but actually avert certain death. Potain's aspirator for the pleural effusion, and Southey's tubes for the ascites or anasarca in the legs, are the instruments generally utilised, their method of employment being described under the treatment of Hydrothorax and Ascites respectively. It has been contended that tapping encourages a further drain from the blood into the cavities or tissues, but the procedure has often saved life and given the over-burdened heart and emunctories a chance of recovering their functioning powers. A dry diet often helps to diminish the œdema.

Cardiac tonics must be administered where rest alone is insufficient. These may be direct or indirect in their

action. The direct group includes digitalis, strophanthus, convallaria majalis and caffein. These all cause slower and fuller, and therefore more regular, cardiac contractions in medicinal doses. The tincture of digitalis may be given in 5 to 15 minim doses, but if pushed it contracts arteries, owing to the action of digitoxin and digitalin, and may therefore add to the work of the heart. The infusion in 1 to 2 drachm doses rather dilates arteries, as it contains more digitonin (which dilates vessels) than the tincture, and is perhaps therefore a better diuretic. Digitalis may be pushed too far, and if so irregular and rapid action of the heart and a drop in the amount of urine passed are likely to occur. Nativelle's granules of digitalin, which really contain mostly digitoxin, are sometimes of value. Strophanthus is not a vasomotor stimulant, and the tincture in 5 to 15 minim doses is often preferred to digitalis, and sometimes carefully pushed by giving 10 minims every two hours for even one or two days it is followed by most excellent results. Both digitalis and strophanthus are apt to cause considerable nausea, especially if there is a tendency to it, and the combination with a carminative, such as 2 to 5 minims of the tincture of capsicum, is most satisfactory. Strophanthin ($\frac{1}{200}$ grain) or digitalin ($\frac{1}{100}$ grain) may be given hypodermically where medicines by the stomach are undesirable. Caffein citrate in 5 to 10 grain doses is also useful. The indirect cardiac tonics include alcohol, the spirit of ammonia, and the spirit of chloroform: often strychnin, arsenic, and iron, singly or in combination, are of great value.

In most cases of increased backward pressure, the passive congestion of the stomach demands careful dietetic and sedative remedies, and engorgement of the lungs with more or less œdema requires stimulating expectorants.

Dyspnœa if due to increased backward pressure may be relieved by the medicinal measures already indicated,

while propping up the head and shoulders gives much comfort to the patient. If, on the other hand, it is due to diminished forward pressure, or, in other words, to anæmia of the brain, diffusible stimulants and opium prove most successful. Cardiac pain and palpitation, generally associated with great enlargement of the left ventricle, may call for the use of opium, the icebag over the heart, nitroglycerine, and sometimes potassium iodide. Sleeplessness occurs both with increased backward pressure and diminished forward pressure, and is often most distressing. Alcohol, the spirit of chloroform, paraldehyde, and in aortic cases opium, give untold comfort to the patient by inducing sleep and allaying restlessness. In considering the treatment to be accorded to the individual valvular lesions, very little need be added to the general account given above. In mitral and tricuspid lesions, increased backward pressure demands special attention; while in aortic insufficiency the cerebral anæmia and the anginous attacks call for treatment. Although digitalis and strophanthus prolong the ventricular diastole, this is no reason why in aortic regurgitation with loss of compensation these remedies should not be prudently used.

SUB-GROUP (δ)—*FUNCTIONAL DISORDERS OF THE HEART.*

FUNCTIONAL HEART DISEASE.

Syn. *Cardiac Neurosis.*

Etiology.—The heart action may be disturbed in many ways and from many causes apart from those already considered, which arise within the heart itself. Any alteration in the nutritional value of the blood will tend

to make itself felt prominently upon the brain and heart, upon the latter not only directly but also indirectly through the brain. Direct action may be upon either the cardiac muscle or its nervous ganglia. Indirect action may arise through a heightened influence of the brain centres or by reflex stimulation by the blood of the afferent nerves of the heart. These local and central causes of disturbance vary in the proportionate influence they exert in different cases. The central are prominent in individuals of a "nervous" temperament. The increased cardiac sensibility to nervous action may, however, arise particularly in nervous individuals in other ways than by the blood path. Reflex stimulation of visceral origin, for instance, may be the cause. Then, again, mere physical or mechanical conditions, such as flatulence, distension of the stomach, or violent muscular effort, may interfere with free cardiac action. The last is well exemplified when men undertake severe muscular work without sufficient preparation, as in the mountaineer trying a difficult ascent before he is in fit training, and also in many army recruits breaking down during training. In most of these cases, if longer time of preparation be given, the heart answers to the increased call made upon it by the steady and more vigorous action it exhibits in the perfectly trained athlete instead of being, as it is earlier, hurried into imperfect and irregular action, causing palpitation, dyspnœa and distress, which makes the work impossible. This is spoken of as the "Irritable Heart."

KINDS OF DISTURBANCE.—It is sometimes one kind of disturbance, sometimes another, or a combination of them, which the functionally upset heart shows. Those most frequently met with are—(1) Palpitation; (2) arrhythmia; (3) tachycardia; (4) bradycardia; (5) pain. Any or all of these may be met with in organic as well as functional disease of the heart, but their presence in the former condition has already been considered.

PALPITATION.

Some observers restrict this term to irregular and excited action of the heart "perceptible to the individual," others apply it also to this action when it is observed by the clinician. These two applications are not the same, for the patient may perceive and complain of palpitation when the clinician cannot detect any irregularity whatever, and the latter may find irregular and excited heart action present, even in a high degree, without the patient being aware of it. It is a symptom very frequently met with from a variety of causes apart from organic cardiac disease, *e.g.*, excited states of the nervous system, as in nervous women, neurasthenics, severe emotions; also in toxic states, such as occur in fevers or after the over-indulgence in tobacco, tea, coffee, alcohol, &c. It manifests itself by quickened, irregular, excitable or more forcible action of the heart of all degrees. The arteries may throb forcibly.

ARRHYTHMIA.

There are many different forms of arrhythmia. (1) The beats during inspiration may be more frequent and full than during expiration. (2) There may be a dropping or intermission of a cardiac beat. (3) A strong heart beat may alternate with a weak one. (4) Non-synchronous action of auricles and ventricles, thus auricular systole may at times occur during ventricular systole instead of diastole. This produces what is known as a group pulse, such as the *pulsus bigeminus* (in which two strong beats are followed by a weak one) or the *pulsus trigeminus* (one beat weak in four). (5) *Delirium cordis*, in which the heart's action is

wholly irregular. These are the chief forms, but others have been described. The extracardiac causes are—(1) Cerebral (psychical influences, concussion, &c.); (2) reflex (visceral disease, &c.); (3) toxic (tea, tobacco, digitalis, &c.).

TACHYCARDIA—RAPID HEART OR HEART-HURRY.

The normal rate of the heart is due largely to the mutually controlling action of the accelerator and depressor centre and nerves. The accelerator fibres leave the cord by the anterior roots of the second and third spinal nerves, and pass to the ganglia stellata and the inferior cervical ganglia of the sympathetic. Thence they reach the heart by the superior, middle and inferior cardiac branches. The depressor fibres come from the nucleus ambiguus in the medulla by the lowest rootlets of the vagus. Excitation of the accelerator nerves increases the frequency and tone of cardiac action, while excitation of the depressor nerves decreases both. Conversely, if either centre be inhibited the other exerts its action to an undue degree. These *disturbing influences of either set of fibres may originate centrally*, as in tumors, hæmorrhages or other affections of the medulla, and in the case of the depressor fibres they may arise reflexly from visceral or peripheral excitation; but there is no evidence that this happens also in the case of the accelerator fibres. The normal rate of the heart varies. The average adult rate is between 70 and 80, but is higher in children, according to their age, thus at four years old it averages 105 to 110. Some adult individuals have a consistently slowly-beating heart—as low as 40 per minute; others have a quick one—as high as 100 or more per minute. A marked disturbance of the individual's normal rate constitutes tachycardia, and it

may arise in a variety of ways. Some of these, both of extra- and intra-cardiac origin, are due to definite lesions in the medulla or in the heart, and can hardly be called neuroses. Others, however, show no discoverable lesion, and may act either by influencing the cardiac centre or the cardiac muscles. Thus fright or other violent emotion acts chiefly upon the centres; while visceral excitation, *e.g.*, the stomach or uterus or ovary, may act upon the vagal centre reflexly. A fall in arterial or a rise in venous blood-pressure increases the heart's action by its influence on the centres in the medulla. A high temperature and the presence of certain toxic substances in blood (particularly the latter) act in the same way and also directly upon the cardiac muscles. Thus the heart's action is increased in most acute fevers, and sometimes from over-indulgence in tea, alcohol, &c. Violent muscular effort increases the heart's action for the same reason; but it is rare for any of these causes to increase the heart's action so much as to constitute a true tachycardia. The best instance of true tachycardia is seen in its paroxysmal form, which is also rare. The patient seems otherwise perfectly well. The paroxysms usually come on suddenly and last for very varying periods—minutes, hours or days. The heart suddenly and greatly increases in rate to, say, 150 or even 200 or more, and may be uncountable. Feelings of distress or dyspnoea may accompany this or may be entirely absent. The paroxysms may come on after violent exercise or spontaneously. They may occur frequently (every week or two) or at rare intervals. They seldom disappear altogether, and sometimes end fatally from heart failure. This peculiar affection has been much discussed, but it still remains very obscure.

Treatment.—Palpitation is often of purely nervous origin, and therefore anxiety should be allayed and the

patient assured that however distressing the symptoms may be there is no danger. Over-exertion of mind, and especially of body, should be prohibited, and a regular life, regular plain diet, and moderate exercise insisted on. Tea, coffee and tobacco must be forbidden, and dyspepsia, flatulence and constipation energetically treated. Turkish and hot baths should not be indulged in. The Weir-Mitchell plan of treatment benefits the neurasthenic type of patient who suffers so often from great abdominal pulsation. Paroxysms of tachycardia are relieved by the icebag, sometimes by aconite, and if accompanied with sleeplessness and excitement by bromides. Not infrequently electrical treatment has proved useful. We have often noted that deep inspirations, raising the arms, and other devices give temporary relief to patients with severe palpitation. Remember *digitalis* may be useful in severe cases not necessarily accompanied by any organic disease; but iron, strychnin and general tonics are most commonly beneficial.

BRADYCARDIA—SLOW HEART.

This name is given to marked habitual or paroxysmal infrequency of the heart. The cardiac beats must be counted by auscultation, as the pulse is not always a safe guide to them. It is not common from either organic or functional mischief. It may arise in the same way as tachycardia—from organic disease of the medulla or heart, and in the latter case more frequently from degenerative myocardial than from valvular lesions. As a neurosis it may appear in altered conditions either of the blood-pressure or of the blood, and thus it is common in convalescence from acute fevers, notably typhoid, acute rheumatism, pneumonia, and diphtheria. This may also be the explanation of its appearance in certain respiratory

troubles, notably emphysema, in which it is not infrequent, and it certainly is the explanation when certain poisons are present in the blood, *e.g.*, lead, alcohol, digitalis and certain specific toxins, &c. It is perhaps most often met with in morbid conditions of the abdominal organs, *e.g.*, chronic dyspepsia, cancer of stomach, jaundice. Increased action of the vagus by reflex stimulation may be its explanation here.

Treatment.—Bradycardia calls for no special treatment: tonics, and especially strychnin, are useful when it follows such diseases as influenza. Occasionally cardiac tonics are necessary.

ANGINA PECTORIS—BREAST PAIN.

This is the most striking of all these affections. Like the others, it is merely the leading symptom, which may arise from many different causes, hence it also constitutes a disease or loosely built up group of diseases having an indefinite clinical but no pathological entity.

Etiology.—In the great majority of cases there is an actual organic lesion present in the heart, *viz.*, sclerosis of the orifices or trunks of the coronary arteries with consequent fatty or fibroid change in the myocardium. There is indeed usually a marked general arterial sclerosis in addition, the patients being almost exclusively males between forty and fifty years of age, and this close association suggests that the sclerosis is the cause of the pain. It does not cause it *per se*, as arterial sclerosis, even of an advanced character, is very common, while angina pectoris is rare; but exactly how it acts is not known. It is thought by some to set up a neuralgia, by others to cause a general or partial spasm of the cardiac muscle, by others to cause an acute dilatation of the

heart and consequent tension on its nerves, while some hold that there is a spasm of the coronary arteries as well. But arterial sclerosis is not the only cardiac lesion found in cases of angina pectoris. Aortic incompetence or adherent pericardium are found next to sclerosis in order of frequency. It is very rare in other valvular lesions, but is occasionally present in acute pericarditis and in tumors of the mediastinum involving the cardiac nerves. In some cases there is a history of gout, diabetes, syphilis, influenza, or over-use of tobacco or alcohol; but in a small proportion of cases no lesion or antecedent disease is discoverable. It is this last group alone which is entitled to be called a neurosis at present. The attacks are spasmodic, hence the lesion or lesions, when present, are not able to induce an attack without help. Attacks come on during exertion, particularly if sudden, or after mental emotion or reflex stimulation from a dilated stomach or a chilled skin. These causes are mentioned in their order of frequency.

Symptoms.—Sudden and severe, even agonising, pain seizes the patient in the region of the heart. It radiates upwards to the neck and shoulder and down the left arm, or occasionally the right, or even both, as far as to the fingers. There is a sense of constriction almost vice-like in the heart region. The patient clings to the nearest support and is afraid to move or breathe. He has a feeling of faintness, of want of breath, and of impending death. The face is usually pale and anxious, and covered with cold perspiration. The paroxysm lasts from several seconds to a minute or two. The pulse may be regular or irregular, frequent or infrequent, of increased or normal tension, but it is often surprisingly little altered even in severe cases. There may be eructations, or the passage of a large quantity of pale urine, or a sudden evacuation of the bowels or stomach after the attack, and the patient may feel some soreness in

the cardiac region, or a general sense of exhaustion for a day or two; but in others he feels quite well soon after. The attacks recur with irregular frequency and at varying intervals. The patient may drop dead during the first or any subsequent attack, or even without warning when not in an attack. In its severe grades it ultimately proves fatal, and this may be soon or after many years. In its milder grades, particularly where no arterial degeneration can be detected and where there is a history of some probable toxic cause, *e.g.*, tea, tobacco, &c., it is not so serious.

Diagnosis.—The pain, the feeling of faintness, and the sense of impending death are the most important factors in the diagnosis. It is simulated by many other varieties of pain, called generally pseudo-angina. Huchard's table showing the differences between true and false angina is very useful.

TRUE ANGINA	FALSE ANGINA.
Most common between the ages of forty and fifty.	At every age, even six years.
Most common in men. Attacks brought on by exertion	Most common in women. Attacks spontaneous.
Attacks—rarely periodical or nocturnal.	Often periodical and nocturnal.
Not associated with other symptoms.	Associated with nervous symptoms.
Vaso-motor form rare (great coldness and stiffness of feet and hands)	Vaso-motor form common. Pain less severe. Feeling of distension.
Agonising pain. Chest feels as if compressed by a vice	
Pain of short duration. Immobility.	Pain lasts one or two hours. Agitation and activity.
Lesions. Sclerosis of coronary artery.	Neuralgia of nerves and cardiac plexus.
Prognosis grave, usually fatal.	Never fatal.
Arterial medication.	Anti-neuralgic medication.

Prognosis.—Few instances of true angina recover completely, though with care and proper treatment the attacks may be diminished in frequency and severity.

Treatment.—A careful study of each case is essential, because much depends on the recognition of any etiological factor present. It must, however, be admitted that the treatment is chiefly palliative, and consists in relieving the paroxysms by the administration of alcohol, the spirit of chloroform or ether, and other antispasmodics. The inhalation of nitrite of amyl, three drops on a handkerchief, is of value in severe cases. Nitrite of amyl acts by suddenly reducing the arterial tension or spasm. Should the arterial tension become habitually high, one drop of a 1 per cent. solution of nitro-glycerine may be given three times a day, and increased until headache indicates that the maximum limit is reached. The drug is particularly useful to prevent rather than to relieve the attack. During the intervals the general health of the patient should be improved; careful dieting must be enforced with not too large meals at one time; and a sufficiency without an excess of exercise enjoined. Heat applied to the cardiac region, whether hot bottle or mustard plaster, will often give much relief during an attack, and in all cases where arterial pressure is high or arterial degeneration suspected, the iodide of potash in 10 to 15 grain doses thrice daily should be given a fair trial. In certain cases, and perhaps especially where aortic incompetence exists, opium should be administered, either in regular doses ($\frac{1}{4}$ to $\frac{1}{2}$ grain of the extract thrice daily) or a hypodermic of morphia ($\frac{1}{4}$ to $\frac{1}{2}$ grain) when an attack of the pain occurs. Patients should lead a quiet life and avoid all excitement and sudden exertion.

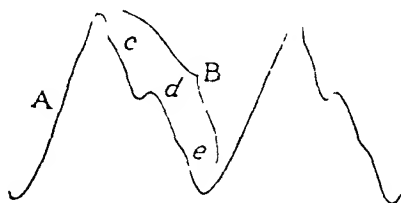
II.—DISEASES OF THE BLOOD-VESSELS.

THE PULSE.

There are two ways of examining the pulse—(1) By the finger, and (2) by instruments. The finger gives the fuller and more reliable information. It can determine—(1) the size; (2) the tension; (3) the state of the walls of the artery; (4) the rate, rhythm, amplitude, duration, dicrotism, force and time of the pulse wave. It is usually felt at the wrist, and a comparison should be made with that of the other side. The size or volume of the pulse may be large, medium, small or thready. It depends immediately upon the amount of blood in the radial artery, the state of its wall and of the heart, and the fulness of the *venæ comites*. The tension is estimated by pressing the finger upon it just enough to obliterate the artery between the beats. We speak of it as high, hard, tense, full or resistant when it is difficult to compress it, and as relaxed, low, soft or empty when it is not. It depends upon cardiac action, the peripheral resistance, and the volume of blood. The state of the walls of the artery is determined by compressing the artery and then rolling it under the finger between it and the radius, and by tracing its course upwards. The rate of the pulse is counted with a watch. The average rate varies with age—*e.g.*, in the *foetus* it is 130 to 140; from birth to one year 130, and gradually comes down to about 100 at five years of age and 90 at ten years. until in adult life it is 70 to 80. It is spoken of as quick, rapid or slow, according as it is much above or below the average. The rate is that of the cardiac pulsations, except in rare instances, when the latter may be considerably more frequent. The rhythm refers to the regularity or irregularity of the pulse in rate, size, force, &c. It is intermittent

when a beat is missed occasionally. Other irregularities, viz., the pulsus bigeminus and pulsus trigeminus, have already been explained. The amplitude is measured partly by sight and partly by touch, viz., the amount that the artery during the pulse raises the finger lightly resting on it. It depends upon the force of the heart and upon the arterial pressure. The duration refers to the length of time the pulse wave takes to pass. It may be quick (*i.e.*, short) or slow (*i.e.*, long). Dicrotism refers to the appreciation of a second beat following upon the primary one. It always indicates a low arterial pressure. The force of the pulse wave is estimated by the strength of the shock received by the finger when pressing firmly upon the artery. The pulse is then spoken of as strong or weak. It depends on the force of the heart, the arterial tension, and the site of the artery, and is not in itself a safe guide as to the strength of the heart. The time of the pulse is estimated by feeling the radial artery with one hand and the apex beat with the other. If a longer interval than usual intervenes the pulse is said to be delayed. This is common in aortic disease and in mediastinal tumors. The time of the two radial pulses should also be compared. The sphygmograph is the commonest of the instruments used in examining the pulse. Dudgeon's is the instrument most frequently used, but most clinicians prefer Marey's. It gives a record of the pulse wave, which shows a primary ascent or wave, due to the blood sent out of the heart by the ventricular systole travelling down the arteries at an average rate of seven to eight miles per second. The momentum of the lever carries it higher than the wall of the artery, and when it falls it encounters the still tense wall of the artery, which throws it up a little again, constituting a small secondary wavelet—the predicrotic wave—and a descent, in which there is a secondary ascent called the dicrotic wave, due to the elastic recoil of the expanded aorta. The predicrotic wavelet may even occur in the ascent and not in the descent, when it is

called *anacrotic*. Such pulse tracings are often very valuable in disease, though their interpretation is frequently not at all easy. A well-marked predicrotic wave is a sign of high tension, and a well marked dicrotic wave of low



A, ascent; *B*, descent, *c*, predicrotic wave, *d*, dicrotic wave, *e*, wave or wavelets which may occur at end of descent

tension, in the pulse. The arterial pressure is measured by the Hill-Barnard sphygmometer or Oliver's hæmodynamometer; while the calibre of the artery is measured by Oliver's arteriometer.

SUB-GROUP (a)—DISEASES OF THE ARTERIES

Atrophy of the arteries is met with either as a congenital defect (hypoplasia) in some chlorotic girls, when the heart, aorta and larger vessels are abnormally small, or as an acquired condition, when it is chiefly local in connection with atrophy of an individual organ or limb.

Hypertrophy of the arteries is met with either in the increase in size of a part or in the establishment of collateral circulation. The arteries get larger and wider, and often twisted.

THE DEGENERATIONS.

Fatty degeneration occurs in the intima of arteries of all sizes. It is common in the aorta as whitish or whitish-yellow spots or streaks. The endothelial cells become crowded with fat globules, and later the process extends to the deeper cells of the intima. Fibrous tissue formation may occur around these areas. In capillaries it may lead to rupture, and cause minute hæmorrhages. Fatty degeneration occurs also independently in the middle coat of arteries, especially in the small arteries of the brain, where it may also lead to rupture. It is caused by changes in the blood, particularly the presence of toxic substances. It is also an important process in atheroma calcification. Deposition of calcareous salts may occur either in the intima or media. In the former position it is usually associated with fatty degeneration or atheroma, in the latter position it may involve large lengths of the vessels, especially the middle sized arteries of the abdomen, converting them into rigid tubes. It may cause senile gangrene in the lower limbs.

Hyaline and waxy degenerations of the arteries have already been noticed. The vessels generally become more permeable to fluids, &c., hence the polyuria in waxy kidney and the diarrhœa in waxy intestines.

Necrosis may affect the vessel wall from the inside by a necrotic embolus or thrombus, or through the vasa from the outside vasorum, or more commonly by the extension of a necrotic process in the tissues around to the wall of the artery, as in tubercle and diphtheria. This is of common occurrence in chronic tuberculous disease of the lungs. The walls of the arteries are invaded, and the vessel in time destroyed.

Hæmorrhage often occurs from this cause, and may be very severe. It would happen in the late stages of chronic

phthisis, in which it is one of the most characteristic signs, much more frequently than it does were it not for a preceding thrombosis sealing the vessel before actual necrosis of its walls takes place.

INFLAMMATION—ARTERITIS.

Etiology.—Many of the same influences which produce degenerations may cause inflammation of the arterial walls. Disease of the surrounding tissues may spread to their walls. Poisons may reach them through the blood-stream, either from their lumen or from the vasa vasorum. Many poisons are credited with acting in this way, *e.g.*, septic, syphilitic, tuberculous, influenzal, typhoid, alcoholic, lead, gouty and diabetic. Embolism and thrombosis are obvious causes. Senility and strain are both held to be important factors, while chronic cardiac and chronic renal disease are frequent forerunners. Their mode of action is not clear. It may be that they act mainly by throwing more work upon the arteries, which then dilate. Thickening of their walls follows, but whether this is a simple compensatory process or an inflammatory one as the result of cellular degeneration is uncertain. Disordered metabolism or renal excretion alike may be responsible for a toxicity of the blood, which in turn may cause degeneration or inflammation or both within the arterial walls. Wounds may implicate the arteries. Heredity also seems to play an important part, as chronic arteritis often runs in families. In whichever coat the inflammation begins it usually spreads to the others, but the terms *periarteritis*, *mesarteritis* and *endarteritis* are used when the main incidence is in the outer, middle or inner coats respectively. The inflammatory process is either acute or chronic.

ACUTE ARTERITIS.

This is common enough as a local, but rare as a general process. The former may cause acute aneurism, or hæmorrhage, or necrosis, particularly when septic embolism is present. The clinical results vary widely with the particular artery involved. The general disease, *i.e.*, where it involves many arteries (those of the legs being most frequent), has been said to follow influenza and typhoid. It causes thrombosis, with consequent coldness, œdema, &c., and even gangrene in the affected limb.

CHRONIC ARTERITIS.

Some authors describe a tuberculous, a syphilitic, a thrombo-proliferous (organisation of a thrombus) and a hæmatogenous arteritis, according to origin, but the process is much the same in all. The inner coat is the one which is mainly affected in many forms of chronic arteritis, hence they are often spoken of as endarteritis. Endarteritis obliterans is used to designate that form of inflammation which tends to obliterate the lumen. It is most frequently met with in small arteries, particularly of the brain in syphilis of the lungs, in chronic phthisis of the coronary arteries of the heart, in arterial sclerosis, and of the kidney in chronic interstitial nephritis. The intima is greatly thickened by a new formation of cellular or fibro-cellular tissues (according to the rapidity of the process), the result of a proliferative inflammation of the cells of the intima following upon a thrombosis, or upon some poison circulating in the blood. In syphilis the intima is greatly thickened in this way, while the vasa vasorum of the adventitia are generally dilated, and there is much cellular

infiltration around them, the media being but little affected. When, on the other hand, the disease spreads from a focus near the vessel, as in tubercle, the coats of the vessel are usually involved in sequence from without inwards. Diffuse arterio-sclerosis or arterio-capillary fibrosis, a most important form of chronic arteritis, is met with in every artery in the body, particularly those of the brain, heart, kidneys and limbs. While the change is widely spread, affecting many arteries at the same time, it generally affects individual arteries in a patchy manner, viz., local whitish or yellowish-white thickening of the intima, circumscribed or diffuse, indistinguishable by the naked eye from those of syphilitic arteritis, and often also from those of atheroma. Many authors, indeed, include atheroma in the term arterial sclerosis, and with reason, as in most cases it leads to sclerosis; but in so far as it frequently differs from diffuse arterial sclerosis, not only in its method of production, but also in its distribution, being much more localised, it is better to keep it separate. Diffuse arterial sclerosis is characteristically seen in older age in cases where the circulation has been subjected to prolonged strain, or in cases of chronic cardiac or renal disease. Its relationship to chronic renal disease has been the subject of much controversy, some writers holding that it is secondary, others primary, to the renal mischief. It includes a variety of arterial changes, capable of being produced in many ways, which come naturally into the two heads of circulatory strain and circulating poison. The strain may be caused centrally (cardiac) or peripherally; while lead, alcohol, syphilis and products of disordered metabolism are held to be the most important among the causative poisons. Cardiac hypertrophy and dilatation, with, later, apoplexy or cardiac failure, are among the most frequent results which may follow upon arterial sclerosis. Periarteritis nodosa is a rare form of chronic arteritis met with in the arteries of the brain, serous membranes, &c.,

which is attributed to syphilis or some other toxin in the blood. It produces small nodules upon the vessels, looking to the naked eye like little aneurisms, due to cellular or fibro-cellular proliferations of the outer or inner or all the coats.

Symptoms.—They are extremely various, depending upon the degree of the change and the organ or tissue affected. They are mainly referable to the consequent anæmia, thrombosis, embolism, or hæmorrhage.

Treatment.—The treatment of chronic arteritis varies greatly with the type of the disease present. Stop alcohol, treat syphilis if present, regulate physical and mental work, and restrict the dietary, especially where overeating is an element in the case. Attend carefully to the emunctories, and especially the bowels. High tension may further be relieved by nitrites or nitroglycerine, and in cases with cyanosis and threatening of cardiac failure venesection may be called for. Potassium iodide has a distinct and almost specific action in arterio-sclerosis, largely perhaps by reducing the intravascular pressure.

ATHEROMA. }

Etiology.—This name is given to a disease of the arteries in which degenerations and proliferative changes are the main features. Two views are held as to its origin. The one regards it as primarily a degeneration, the other as primarily an inflammation of the inner coat occurring either through circulatory strain or a circulating poison, and is therefore most often seen in persons of advanced years (particularly after fifty) who follow laborious occupations or those who are the victims of chronic alcoholism, gout,

syphilis, plumbism, and probably as a sequel to acute arteritis caused by some acute infectious process, such as typhoid, scarlet fever, &c.

Morbid Anatomy.—It affects arteries of all sizes in the aorta, it is most marked in the arch, particularly near the orifices of its branches, viz, the coronary, innominate, left common cardiac and left subclavian arteries. The changes may be arranged in three stages. In the early stage there are translucent or whitish circumscribed moderately firm elevations of the inner coat. They vary in size from minute foci to others much larger than a split pea. They have a small surface and tend to be somewhat circular in outline, but later form very irregular areas from influence. In the second stage fatty degeneration sets in in the deeper and central parts of each focus, giving it a yellowish colour. This extends until it involves the greater part of each patch. When more rapid and complete, some of the patches may become soft and pultaceous and are sometimes spoken of as “atheromatous abscesses.” They may break through the surface of the intima and discharge their contents into the blood-stream, leaving an ulcer. In the third stage calcification occurs in the fatty patches and may lead to the formation of calcareous plates of some size, which are flat and depressed, particularly in their centres, though their margins may be raised and may even break through the intima. Thrombosis may take place over them or over the “atheromatous ulcer,” or the blood may force its way beneath them and form a new channel for itself within the coats of the aorta (dissecting aneurism). The most common result is dilatation of the lumen of the aorta, which may be diffused or localised and saccular (aneurism). The microscope shows that the changes in atheroma begin in the deeper parts of the intima, next to the media. They are constituted by, firstly, a proliferation of the cells and then of the fibres, followed by fatty

degeneration and, later, calcification. There is also much swelling and hyaline change in the fibres. The tissues break down, forming irregular spaces partially filled with degenerated detritus, fatty matter, cholesterin, &c. The process extends inwards to but does not involve the endothelium. It sometimes involves the media but rarely the adventitia.

Symptoms.—The most important signs are an increased area of dulness over the manubrium, a rough systolic aortic murmur replacing the first cardiac sound, hypertrophy of the left ventricle, and an ample, slow, often irregular radial pulse.

Atheroma affects the pulmonary and other large arteries in the same manner as the aorta. It is often present at the same time in the medium-sized arteries like the radial, or may occur there independently. It forms local whitish thickenings of the intima, similar to those already described under syphilis and arterio-sclerosis. These thickenings bulge irregularly into the lumen, giving the transverse section of the artery an appearance which has been likened to a signet ring.

Treatment.—All that is requisite is to keep in remembrance that degenerated arteries cannot resist strain satisfactorily, and therefore exercise should be proportionate to the condition of the vessels. Further, the bowels should be kept freely open and the amount of alcohol permitted ought to be the minimum amount required.

ANEURISM.

DEFINITION.—A localised enlargement of the whole lumen, or a saccular projection from one part of the lumen of an artery.

Forms.—There are several varieties of aneurism, but the most important are *the true*, in which the wall of the aneurism is one or other of the arterial coats; *the false*, in which it is formed by the condensed tissues around after rupture of the wall; and *the dissecting*, in which the blood ruptures the inner coat and makes a new channel for itself within the coats, generally within the substance of the media or between the adventitia and the media.

Etiology.—Any process which brings about a weakening of the arterial wall may result in aneurism. Thus atheroma and sclerosis generally cause loss of elasticity in the affected parts of the arterial wall. The elastic fibres of the media are found to have disappeared, and the pressure within will cause dilatation of the lumen owing to the loss of resistance in the wall, particularly if the proliferative changes in the intima in the sclerosed vessel are of slow formation and not strong enough to compensate for the loss of elasticity. Actual rents or tears may occur in any of the three coats. Circulatory strain and circulating poisons are thus, as in sclerosis, the primary causal factors. Embolism may result in an aneurism on the proximal side of the embolus, particularly when the latter is septic.

Morbid Anatomy.—The thoracic aorta is the most frequent seat of aneurism, but it is not uncommon in the abdominal aorta or its branches—the popliteal, the carotid and cerebral arteries among others. It is either fusiform, cylindrical or saccular in shape, and varies greatly in size. Its cavity is filled with liquid or a mixture of liquid and coagulated blood. The coagulum may be red, but in saccular

aneurisms it is more often white, and consists of layers of fibrin often distinctly laminated, like the layers of an onion ; but true organisation is very rare. The inner layer of the wall of the sac is smooth and frequently extensively atheromatous. The intima is usually only discernible at intervals. The media has likewise disappeared, although traces of it may be got at the mouth of the sac. The adventitia is greatly thickened, and along with the condensed surrounding tissues forms the wall of the aneurism. The changes found in other organs and tissues depend chiefly upon the site of the aneurism. They are mostly pressure effects which lead to irritation, atrophy, erosion, and absorption. Soft tissues resist the pressure of the increasing aneurism better than hard ones, thus bone is more affected than cartilage, and cartilage than soft fibrous tissues. It erodes the bodies of the vertebræ more quickly and extensively than the intervertebral discs, the sternum more than the subcutaneous tissues. It may disturb tubes of all kinds, such as blood-vessels, especially veins, lymphatics, the œsophagus, bronchi, &c. It may irritate or paralyse nerves. When near the heart, it may cause hypertrophy of the left ventricle, and, finally, it may be cured spontaneously by its cavity being completely filled by thrombosis, or it may prove fatal by rupture into some adjoining cavity or viscus or through the skin.

Symptoms.—These are mainly due to its pressure effects, and will depend largely upon the anatomical position of the aneurism. If there be no pressure effects the aneurism will be latent and may not be discovered at all, or only accidentally, or when they rupture. Usually there are more or less well-marked symptoms and physical signs, and those of aneurism of the thoracic aorta may be taken as a good illustrative example. The most important symptoms are—(1) Pain ; (2) cough and expectoration ; (3) dyspnœa ; (4) alterations in the voice ; (5) dysphagia ; and (6) hæmoptysis.

The pain is very variable. It is most often present in deep-seated aneurisms. It is of two kinds—permanent and intermittent. The first is referred to a particular spot, and is usually increased by exertion and at night. The second is neuralgic or even anginal in character and felt along the courses or at the terminations of the brachial or intercostal nerves. The cough may be moderate and occasional, with copious watery expectoration diminishing in amount and becoming thick as time goes on, or it may be paroxysmal and severe, and of a peculiar brassy ringing or clanging character. The former is due mostly to direct pressure on the windpipe, the latter to pressure upon the left recurrent laryngeal nerve. The dyspnœa may be present only after exertion, or it may be paroxysmal and spontaneous, and due to pressure on the windpipe or on the recurrent laryngeal nerves. The voice may be hoarse or lost, due to pressure on the laryngeal nerves, particularly the left, inducing spasms or paralysis of the muscles of the left vocal cord. Dysphagia is a rare symptom, being met with occasionally in aneurisms of the descending part of the arch and the aorta pressing upon the œsophagus. An œsophageal bougie should never be passed where an aneurism is suspected, as fatal rupture may follow. Interference with the first stage of swallowing, viz., the passage of the food over the larynx, is more common than true dysphagia, and may cause dyspnœa and cough during meals, or even septic bronchopneumonia. Hæmoptysis may be slight, the sputum being merely tinged with blood, or small quantities of pure blood may come up persistently for long periods. This is caused by moderate leakage into the pulmonary alveoli or air passages. At other times the bleeding may be profuse and rapidly fatal, from rupture into the lung or air passages. Fatal hæmorrhage may take place also into the œsophagus, pleural or pericardial cavities, or elsewhere. Profuse hæmorrhage may occur and recur, and the patient recover and live for years.

PHYSICAL SIGNS.—Inspection should be made by looking obliquely at the chest in a good light. It may show pulsation or a pulsating swelling to the right of the sternum in the first or second interspace when the first part of the arch is involved, in the supra-sternal notch when the transverse part is affected, or it may occupy the upper part of the sternum when the latter has been absorbed. When seen posteriorly it is usually in the left scapular region. Inequality of the pupils may be observed from pressure on the sympathetic on the upper part of the chest. Palpation determines more accurately the area and degree of pulsation. It should be made after a full expiration in the stooping posture. When the aneurism is large and has perforated the chest wall the pulsation is heaving and *expansive* and systolic in time, followed by a diastolic shock, often most pronounced. This systolic and diastolic shock are frequent and important signs of aneurism. A systolic thrill is also present at times. There may be also œdema of the arm or back, or both, and dilatation of the superficial veins, from pressure upon the superior vena cava, and the aneurism may even rupture into it. The pulse in the arteries beyond the aneurism may be slower; thus there may be a marked difference, both in volume and time, between the radial pulses of the two sides. Tracheal tugging may be present, particularly in deep-seated aneurism of the highest part of the arch. It is manifested by the clinician feeling a gentle tugging on the trachea downwards, synchronous with each cardiac systole, when he presses the cricoid cartilage lightly and steadily upwards. Percussion yields negative results except when the aneurism is of some size and is in contact with the chest wall. Such an aneurism of the ascending arch gives a dull area to the right of the sternum, of the transverse arch to the mid part of the manubrium and extending towards the left, while those of the descending arch and aorta give dulness in the left interscapular or scapular regions. Auscultation may

detect a systolic murmur, or a diastolic, or both. The first is of either aneurismal or cardiac (valvular) origin, while the second is always valvular (aortic incompetence), and hence neither of them are reliable signs. Of far greater importance is the character of the second cardiac sound—the diastolic shock sound as it is called when it is heard over the area of the aneurism. It is loud and ringing or booming. There may, further, be physical signs of pulmonary collapse or of pleural effusion.

Prognosis.—It is always grave, but the degree of gravity depends mainly upon the rapidity and direction of growth of the aneurism and upon the circumstances of the patient. Generally those which grow backwards cause more severe symptoms and greater danger than those which grow forwards, even though the latter may form large tumors. It is generally a chronic disease of indefinite duration (often several and sometimes many years), though sudden death from rupture or cardiac failure may occur at any time.

Treatment.—The medical treatment of an aneurism may be considered under three closely associated heads—relief of symptoms, attempt at what is called cure of the aneurism, and the adoption of measures to prevent existing dilatations of the vessel from becoming worse. Absolute rest in bed should be enjoined so as to reduce the blood-pressure, all physical effort must be given up, the bowels kept freely open, straining at stool being specially detrimental, and a carefully regulated diet enforced. Whether we advocate Tuffnell's starvation method of treatment or not, his principle of a minimum amount of food, and especially fluids, is a most beneficial one. By lowering the blood-pressure we hope to get thrombosis to occur in at least saccular aneurisms, and also to ensure that the distressing symptoms of the aneurism will be alleviated. Tuffnell's dietary

consists in—for breakfast, 2 oz. of bread and butter and 2 oz. of milk; for dinner, 3 oz. of meat and 3 or 4 oz. of milk or other similar fluid; and for supper a repetition of the breakfast diet. The great drug for lowering pressure is potassium iodide: George Balfour gave it in small doses, 10 to 15 grains thrice daily, other physicians push the dose up to 60 or 90 grains thrice in the day. By its use pulsation becomes much less marked and pain is greatly alleviated.

Several surgical procedures have been recommended to aid clotting, such as the introduction of fine gold wire or even horse-hair into the sac; scratching the inner wall of the sac to induce clotting; electrolysis; and, more lately, gelatine (100 ccm. of a 2 per cent. solution) injected, preferably by the skin or into the bowels, has been found successful, in some cases fifteen to twenty injections are generally requisite, and the gelatine should be rendered sterile before use. When an aneurism bulges externally and threatens to burst, an icebag should be applied, or belladonna plaster or collodion used as a support to the skin. Opium relieves pain and dyspnœa when potassium iodide together with rest in bed fail to sooth the sufferer. Venesection may sometimes be of advantage, where, for example, the superior cava is pressed on. Continuous pressure upon the sac of an abdominal aneurism when in a suitable position (associated of course with bed, low diet and potassium iodide) is indeed Spartan treatment, and the pain of the process calls for morphia. One difficulty in treating any aneurism—thoracic or abdominal—is that we can never feel certain when the maximum amount of cure has been obtained, and not a few patients prefer to die in harness, merely taking the precautions already indicated to avoid any special increase of intravascular pressure.

SUB-GROUP (β)—DISEASES OF THE
VEINS.

These have already been considered under various headings, *vide* Varicose Veins, Thrombosis, Phlebitis. A chronic inflammatory change similar to arteriosclerosis, and often spoken of as phlebosclerosis, occurs in the veins. It occurs along with arteriosclerosis and depends upon the same causes. Acute phlebitis is very common. It may be caused by traumatism or by extension of a neighbouring inflammation or by germs or poisons circulating in the blood. Further description of these conditions is not required in a book of this size.

Section 5.

DISEASES OF THE RESPIRATORY SYSTEM.

It is computed that, even if pulmonary tuberculosis be excluded, 30 per cent. of all the graver cases of illness which come under the physician's care are disorders of the respiratory system. The mortality from respiratory disease is greatest at the two extremes of life; but even over all ages it reaches the high figure of 20 per cent. if pulmonary tuberculosis be excluded, and 28·5 if it be included, of the deaths from all causes. It is convenient to divide respiratory diseases into those of the nose, larynx, bronchi, lungs and pleura.

I.—DISEASES OF THE NOSE.

ACUTE NASAL CATARRH.

Syn. *Acute Coryza*, *Acute Rhinitis*.

DEFINITION—An acute inflammation of the mucous membrane of the nose and usually also upper air passages (above the larynx), generally spoken of as a "cold" or a "cold in the head."

Etiology.—While common enough at all ages and at all periods of the year, it is most common in young people and in the changeable cold weather of winter and spring. A chill from sitting in a draught or from getting wet

frequently precedes an attack. Certain individuals are more susceptible than others, and this increased susceptibility may be marked in different members of the same family. But age, season of the year, chill and heredity, however important their influence, act only as predisposing causes, the real exciting cause being a micro-organism. No specific micro-organism has yet been discovered, but various bacteria, particularly the pyogenic cocci, have been found in the nasal secretion, and it may be that any one of them, gaining entrance to the suitably prepared soil, can set up an attack, or it may be a specific organism as yet unknown, and the sudden onset, course and contagiousness of the disease rather support this view. Irritating fumes—*e.g.*, bromine or ammonia—may cause an acute catarrh of the nose, which disappears with the removal of the cause.

Morbid Anatomy.—The mucous membrane of the nose is congested and swollen. The secretion is increased. It is at first thin and scanty but soon becomes copious. It is irritating to the nasal apertures and upper lip. Herpetic vesicles may appear upon the lip.

Symptoms.—Frequent sneezing is one of the earliest symptoms. There is usually also some headache and a feeling of indisposition. The temperature may be raised (about 101° F.). The nose feels hot and “stuffed up,” necessitating breathing by the mouth. The nasal discharge, at first slight, soon becomes copious and watery. The edges of the nose and the upper lip become sore. The eyes may become suffused. There is often a sense of fullness or of tightness or pain over the frontal sinuses, probably from the inflammation extending to them. The sense of smell is lost, and there may be deafness from implication of the Eustachian tube. The throat may become sore, and the voice is often thick and husky.

The acute stage usually begins to subside within thirty-six hours, the swelling becoming less and the nasal discharge becoming turbid, more profuse, and then diminishing. Recovery is generally complete within four or five days.

Diagnosis.—The early stage of measles and influenza resembles a common cold, but the distinction is generally easily made.

Treatment.—In many ways a cold in the head may be aborted if treated promptly. A hot bath, the free use of such a diaphoretic as the liquor ammoniæ acetatis, a saline or other purgative, and at bedtime a single dose of opium in the form of 10 or more grains of the compound ipecacuanha powder are all excellent. Locally, painting the nasal mucosa with a 5 per cent. solution of cocain, applying on pledgets of cotton-wool or by spray one of the preparations of suprarenal gland, or using a snuff, such as Dr Ferrier's (morphia, acacia gum and bismuth), may frequently give much relief to the patient. When a case has reached an advanced stage and is very severe, expedite recovery by confining the patient to one room and giving steam inhalations.

CHRONIC NASAL CATARRH.

This is an extremely troublesome condition. Several varieties are described, *e.g.*, simple, hypertrophic, atrophic, and the changes they set up are indicated by these names. The association of the hypertrophic form with adenoid is common. The chief symptom of them all is obstruction to the natural method of breathing, viz., through the nose, and a consequent resort to mouth breathing, so that the mouth and throat get dry during sleep.

Treatment.—Much depends on the nature of the condition present. Merely a chronic coryza may yield to change of air and tonics ; but if there is much long-standing hyperæmia of the turbinateds, the galvano-cautery may be necessary, especially if such astringents as alum and silver nitrate fail to give relief. Where there is the slightest tendency to a foetid discharge, the nose must be douched with antiseptics, such as boric acid, listerine, &c.

In both acute and chronic nasal catarrh the most careful attention should be paid to the state of the Eustachian tubes, and if any degree of deafness be present the middle ear should be inflated.

HAY FEVER.

Syn. *Hay Asthma, Autumnal Catarrh.*

DEFINITION.—An affection of the upper respiratory passages, characterised by paroxysmal attacks of nasal catarrh recurring every summer or autumn.

Etiology.—It is now recognised that three factors play a part in the causation of this disease, viz., a nervous constitution, an irritable or abnormal nasal mucosa, and the presence of some exciting stimulus, and some combination of these is probably required in every case.

I. THE NERVOUS CONSTITUTION.—It is mainly, if not exclusively, met with among the educated classes of civilised peoples (Anglo-Saxons, French and Germans), and often runs in families, whose different members, moreover, are likely to show other disorders of a nervous character, such as asthma, chorea, or epilepsy. Mackenzie induced an attack by offering a susceptible person an artificial rose to smell. Dwellers in cities are more liable

to it than those who live in the country. Men are, however, more liable than women, and only a small proportion of the total number of neurotics suffer from it.

2. ABNORMALITIES OF THE NASAL MUCOSA.—Polypi, adenoids, septal or turbinated irregularities, hypertrophic rhinitis and hypersensitive areas are all more or less common, and a correction of the abnormality is frequently followed by a cure. In many cases no abnormality can be discovered, and similar abnormalities are often present in those who never suffer from hay fever.

3. THE EXCITING STIMULUS.—The pollen of certain flowering grasses, various kinds of dust or perfumes, emanations from animals, particularly cats, have all been shown to cause an attack in susceptible persons, and removal of the stimulus, or of the individual from the region where the stimulus prevails, is often followed by cessation of the symptoms. The disease is therefore most prevalent during the summer and autumn months—June to September.

Morbid Anatomy.—In addition to the abnormalities which, as already mentioned, may be present, there is, during an attack, swelling and congestion of the mucosa of the inferior and middle turbinated bones and a free serous discharge.

Symptoms.—They resemble those of an ordinary severe coryza. Sneezing is generally very troublesome and headaches severe, but there is no fever. There may be distressing cough and much depression of spirits. The duration and severity of the attacks vary. The paroxysms recur more or less frequently during the summer months, and often terminate as abruptly as they began. Attacks of bronchial asthma may accompany or replace or alternate with them.

Treatment.—Careful attention must be paid to any of the three etiological factors referred to, and which may be present. The nervous element must be combated, any nasal abnormality treated, and the patient sent away from regions where hay pollen—the usual cause—is plentiful in the air at certain seasons. A sea voyage is the ideal method of carrying out this last direction, the seaside is next most efficacious. Tonics are of value, and the patient should be specially fortified before the hay season comes round. Probably cauterization has given more relief locally than anything else. Many local preparations—anæsthetic and astringent—are used, such as carbolic acid, 10 to 20 grains to the ounce of paroline, painted on the nasal mucosæ, or menthol solutions of varying strengths. The eyes may require bathing with a weak boric lotion, and one of the antipyrin group of remedies or hydrobromic acid may be given to relieve the headache.

EPISTAXIS.

DEFINITION—Bleeding from the nose.

Etiology.—It is most common about the age of puberty, but it may occur at all ages. It occurs from many causes, which may be classified as local and constitutional. The local causes are due either to injury or disease. Blows upon the nose, fracture of the nasal bones or of the anterior fossa of the base of the skull are examples of the former, and inflammations, ulcerations and tumors are illustrations of the latter. Among the constitutional causes may be mentioned the plethoric habit, certain infectious fevers, particularly typhoid, hæmophilia, chronic anæmias, chronic valvular disease of

the heart, balloon and mountain ascents, chronic liver affections, notably cirrhosis. It occurs with great frequency in young adolescents without any discoverable cause.

Symptoms.—The blood comes from one more often than both nostrils. It may drip or run in a continuous stream. It may be slight or very great; but death therefrom is rare. "A spot about half-an-inch from the anterior end of the cartilaginous septum" is called the "site of predilection" because of the frequency of its being the bleeding spot.

Diagnosis.—The nose may not be the true site of the bleeding, but only its channel of escape, as in fracture of the base of the skull. Again, the blood may flow backwards into the throat and excite vomiting or coughing and simulate hæmatemesis or hæmoptysis.

Treatment.—A slight attack of epistaxis will probably require no more energetic treatment than the popular remedies, such as laying the patient flat on his back, keeping the arms raised, or applying something cold to the nape of the neck, such as a large key. If the bleeding is severe, the head should be slightly inclined forward, so as to let the blood escape by the nostrils, and a pledget of cotton-wool soaked in adrenalin or the tincture of the perchloride of iron should be introduced into the nostril. The posterior as well as the anterior nares may need to be plugged, and in this case the pledgets used should, if possible, have a little iodoform or other suitable antiseptic sprinkled on them, for they may need to remain *in situ* for some time. In every severe case study any constitutional factors present, and give tonics or other suitable treatment where necessary.

ADENOIDS.

Syn. *Post-nasal Growths, Adenoid Vegetation, Hypertrophy of the Pharyngeal Tonsils.*

DEFINITION—A hypertrophy of the lymphoid tissue of the roof of the pharynx.

Etiology.—They are most common between the ages of five and fifteen, rare in adults, and occasionally met with in infants. The true cause is unknown; but repeated catarrhs, bad hygiene, and certain specific fevers—*e.g.*, measles and scarlet fever—have been blamed. Certain cases of secondary syphilis, lymphatic leukaemia, and of cleft palate show large adenoids.

Morbid Anatomy.—They usually form smooth, rounded, reddish masses, varying in size from a small pin head to that of a pea, or sometimes as large as a hazel nut. At other times they form a more regular cushion-like swelling. They consist of lymphoid tissue exactly like that of the tonsils, covered on their free surface by a single layer of ciliated columnar epithelium.

Symptoms.—There may be none, or such slight ones as to escape notice. Usually the nose is obstructed, which induces "mouth breathing," the mouth and throat become dry, and snoring and disturbed sleep supervene. There is a constant tendency to "snuffling" or "hawking," and there may be a harsh, dry, "barking" cough, often called a "stomach cough." The mouth is kept half open and the face usually assumes a dull vacuous look. Deafness is common. It is due either to direct obstruction or to catarrh of the Eustachian tubes. The voice acquires a "dead" quality, from interference with the resonating qualities of the naso-pharynx. Respiratory catarrhs become common.

Treatment is mostly surgical. If adenoids are giving trouble they must be removed. Tonics, such as cod-liver oil and iron, are useful, both before and after the operation.

II.—DISEASES OF THE LARYNX.

ACUTE CATARRHAL LARYNGITIS.

Etiology.—Injury to the larynx, whether mechanical or by corrosive or hot liquids, is rapidly followed by inflammation. Irritating gases, cold, and overuse of the voice appear similarly to excite it. It may arise also from some infectious disease, *e.g.*, scarlet fever. These and many other agents are predisposing, even if they be not exciting, causes, though it may be that in all cases there is a direct exciting cause in the form of a germ. There is, in all probability, no specific germ, but only one or other of the germs of ordinary inflammation. The inflammation may spread to the larynx from the nose or throat, as in measles, influenza and ordinary coryza.

Morbid Anatomy.—The true cords are reddened and slightly swollen. Small superficial ulcers may be seen at their anterior or posterior ends, but they are not common. The aryteno-epiglottidean folds and neighbouring mucous membrane may be somewhat swollen, but marked swelling is rare.

Symptoms.—There is at first a feeling of dryness or a tickling or pinching sensation in the larynx. A dry irritable cough comes on and the voice becomes husky, hoarse and may be painful. Aphonia often follows. There

may be discomfort in breathing and in swallowing, but dyspnœa is seldom present except in children, in whom it may be spasmodic. In severe cases there may be some fever and other constitutional symptoms.

Diagnosis.—In adults a laryngoscopic examination makes the case clear; but in young children, when this cannot be made, the diagnosis is often difficult. Even with the help of a bacteriological examination it cannot always be distinguished from membranous laryngitis, and it is best to treat it as such, in young children at any rate, until the differentiation can be made.

Treatment.—Place the patient in a warm room kept at a steady temperature, and with the air moistened by steam from a steam-kettle. Medication to the larynx may be administered by inhalation, such as the compound tincture of benzoin added to the kettle and inhaled, or by sprays of menthol in paroleine (5 to 10 per cent.) or ipecacuanha wine. Warm baths and poultices will be found of value in certain cases, and a saline purge preceded by a dose of calomel is good routine practice. Remember rest to the voice is imperative. Where asphyxia is threatening, do not delay intubation or tracheotomy till the last moment.

ŒDEMA GLOTTIDIS.

Etiology.—This is a serious, though happily not a common, affection. It is rare in either acute or chronic catarrh of the larynx. Tubercle and syphilis are the two chronic lesions in which it oftenest occurs. It is most apt to appear in severe inflammations of the throat and neck, such as diphtheria, erysipelas, and deep-seated suppurations. It is usually acute, and may come on

very rapidly, as in Bright's disease. In other cases, as in heart disease, it may come on very slowly.

Morbid Anatomy.—The aryteno-epiglottidean folds are greatly swollen, and the cushion of the epiglottis also, though generally to a less extent. The mucous membrane is pale, though there may be congestion at the borders of the swelling. The œdema is usually general, though sometimes it is unilateral, and there is a form which affects the mucous membrane below the true cords as two red fleshy-looking swellings.

Symptoms.—It usually develops rapidly, causing quickly-increasing dyspnœa. There is pain on swallowing, and the voice is husky or lost. The dyspnœa is most serious, and generally causes death. The swollen epiglottis can be felt with the finger. On laryngoscopic examination it may prevent a view of the rest of the larynx, though usually the aryteno-epiglottidean folds are seen, and may even appear to meet in the middle line.

Treatment.—Prompt relief must be obtained. Try scarifying the epiglottis with a curved bistoury if time permits spraying with cocain (5 per cent.) beforehand. Intubation or tracheotomy must be resorted to *at once* if this simpler treatment gives no relief. Where œdema threatens, the application of ice locally to the neck, and lumps of ice to suck, may be tried.

CHRONIC LARYNGITIS.

Etiology.—It may follow the acute or come on independently in those who overuse the voice or who overindulge in alcohol or tobacco.

Morbid Anatomy.—There is swelling and some congestion of the vocal cords and of the interarytenoid fold. There is seldom ulceration. The term “pachydermia laryngis” is employed when the thickening is very marked, and a variety of this is known as “singer’s or teacher’s node,” in which there are small, round, hard nodules on the free border of one or both cords. In addition to this common or hypertrophic form of laryngitis, there are two other rare varieties—the laryngitis sicca, in which the mucous membrane is atrophied, and a glandular variety, in which the minute glands are enlarged.

Symptoms.—The voice is hoarse and rough, and there is usually a tickling sensation in the larynx and frequent cough. There are two forms of chronic laryngitis, viz., the tuberculous and syphilitic, which usually receive separate descriptions.

Treatment.—Caution the patient to rest the voice and to avoid alcohol, tobacco and strongly-spiced foods, especially where it seems probable that excess in one or other of these has been indulged in. Many methods of local treatment are of value, such as inhalations of ammonium chloride; sprays of pine oil, eucalyptus, menthol in paroline; pigments applied with a brush, such as silver nitrate (10 to 30 grains to the ounce), solution of alum (10 grains to the ounce), zinc chloride (5 to 10 grains to the ounce); insufflations of bismuth and other powders, soothing or astringent. A change of air is in all cases desirable, especially where the voice has been overstrained.

TUBERCULAR LARYNGITIS.

Etiology.—It is rare as a primary condition but common as a secondary one following upon tuberculosis of the lungs.

Morbid Anatomy.—An early and frequent change is pallor of the mucous membrane. There is swelling of the cords, of the interarytenoid fold, and of the base of the epiglottis. Tubercular granulations appear in some of these situations, and then ulcers, at first superficial but later becoming deep, invading and destroying the cords, the epiglottis, and even the cartilages of the larynx. Cicatrisation and stenosis of the larynx is a rare result.

Symptoms.—The most characteristic early symptom is huskiness of the voice. It gives place in time to hoarseness and even aphonia. There is cough, and in the later stages, when ulceration involves the epiglottis or pharynx, there is dysphagia, which is often very distressing.

Treatment.—Try to keep the ulcers, if present, thoroughly cleansed. Many of the modes of treating chronic laryngitis are applicable here. Cocain (4 per cent. solution) used as a spray often helps the patient to swallow his food, and ice to suck serves for the same purpose. Some specialists use lactic acid (20 per cent.) applied locally to the diseased tissues, others prefer iodoform insufflations.

SYPHILITIC LARYNGITIS.

Etiology.—It is common both in the hereditary and acquired forms of the disease.

Morbid Anatomy.—The superficial symmetrical ulcers usually affecting the cords of secondary syphilis are not of so much importance as the deeper distinctive lesions of both inherited and tertiary syphilis. Gummata of the size of a small pin's head or larger, up to that of a bean, form within the mucous membrane. The base of the epiglottis is the commonest seat. They usually break down and form ulcers. A single ulcer is much more common than in tubercle, in which they are generally multiple; but the ulceration may be deep and destructive, even more so than in tubercle, as great even as in malignant disease. Cicatrisation may come on early or follow ulceration, and the resulting deformity and stenosis are very characteristic of syphilis.

Symptoms.—The voice is rough and the cough troublesome, but pain and difficulty in swallowing are much rarer than in tubercle. Other symptoms of syphilis are generally present.

Treatment.—The usual remedies—mercury and iodide—must be administered. Remember the rapid and disastrous results of tertiary syphilis when the larynx is attacked, and be prepared for tracheotomy. Constriction of the glottis often follows the cicatrisation, and may need dilation.

TUMORS OF THE LARYNX.

Among the benign tumors, the papilloma is the commonest. It forms a whitish-grey, pink or red warty growth on the vocal cords or anterior commissure most frequently; but while it may be found elsewhere it is never seen on the interarytenoid fold. The fibroma also grows

from the vocal cords as a whitish or reddish nodule of small size or as large as a bean. Among the malignant growths, carcinoma is far more common than sarcoma. Most of them start in the vocal cords. When they arise there or in the ventricles or subglottic region they are called intrinsic, when arising elsewhere, such as the epiglottis, arytenoids, &c., they are called extrinsic.

Symptoms.—Alterations in the voice, dyspnoea and cough are the early symptoms. Pain and dysphagia, &c., come on later in malignant disease.

Treatment.—The treatment depends much on the nature of the tumor. Operative interference is frequently called for.

SPASMODIC LARYNGITIS.

Syn. *Laryngismus Stridulus*, *Spasmodic Croup*.

Etiology.—It attacks young children who are the subjects of rickets, and is a purely nervous affection, there being no morbid change in the larynx.

Symptoms.—The attacks come on suddenly, either during the day or night. The child, who may be just awakening from sleep or may be playing with companions or toys, suddenly struggles for breath and gets blue in the face, due to a spasm of the adductor muscles of the larynx. Presently the spasm relaxes and air is drawn into the lungs with a high-pitched crowing sound, hence the name of "child crowing" by which the disease is sometimes known. The child may be as well as ever immediately the attack is over. Death during an attack is very rare but the attacks may follow one another with great frequency.

Treatment.—The treatment is really twofold—relief of the spasm and an attempt at removing the cause. A hot bath, hot sponge over the chest, smelling salts, dashing cold water over the chest, and sometimes a whiff of chloroform are all good methods of relieving the attack, especially the first two mentioned. A careful study of the case may help to reveal the cause; teething troubles, rickets, and many other conditions which are met with in delicate children predispose. Cod-liver oil, tonics of all kinds and a seaside change of air often work wonders.

III.—*DISEASES OF THE BRONCHI.*

These are acute and chronic bronchitis (medium and larger-sized bronchi and trachea), plastic bronchitis and bronchiectasis. Two others, viz., capillary bronchitis and asthma, are more than mere bronchitic affections, but it is convenient to describe them here.

ACUTE BRONCHITIS.

Etiology.—The causation is much the same as in acute laryngitis. No specific organism has yet been discovered, but many different germs have been found, both in primary and secondary cases, viz., staphylococci, streptococci, pneumococci, the influenza bacillus, &c. There seems to be little doubt that pneumococcal, influenzal, &c., forms of acute bronchitis occur without other sign of disease. They may thus be regarded as primary, but whether there is also a specific primary form caused by some unknown organism we cannot say. Secondary forms

are frequent. They are due to an extension of the cause of the primary disease or to some complicating inflammatory organism.

Morbid Anatomy.—In mild forms the inflammation is confined to the trachea and its primary branches. In more severe forms it affects the bronchi (large or medium sized, or both) throughout the lung. The mucous membrane is swollen and injected, particularly in the larger tubes. The injection and swelling largely subside after death and very little morbid change is visible microscopically at the autopsy. Microscopically, however, there is always some desquamation of the ciliated epithelium, its place being taken by rounded cells, swelling of the homogeneous basement membrane and of the muscularis mucosæ, dilatation of the blood-vessels of the submucous coat, and a general leucocyte invasion of all the coats. The mucous glands are swollen and granular. The lumen of the bronchi is more or less filled with mucus impregnated with cells (leucocytes and desquamated epithelium), but it rarely becomes blocked or dilated except in the smaller tubes.

Symptoms.—Cough and expectoration are the chief symptoms. The former is always present, and it may be severe and hacking, even when there is little expectoration throughout, as in the milder cases. At first it is dry, but in ordinary cases when the bronchi are affected expectoration appears and gradually increases. It is scanty and consists of viscid mucus, but in a few days it becomes more abundant, watery and frothy, often called the sputum crudum. It contains few cells, and these are mostly desquamated epithelial cells. Later, it gradually changes its character, becoming muco-purulent, thick and opaque, from a great admixture with leucocytes, and is now often called the sputum coctum. It soon begins to come up more easily, and the cough and other symptoms, if any, gradually

improve. Dyspnoea is among the most frequent of these other symptoms, but it varies greatly in degree and character, not only with the severity of the disease but with the individual attacked. In some it is very slight, usually showing itself only as a slightly quickened respiration. It is usually more severe in children than in adults, showing not only quickening but difficulty in breathing. Pain is another symptom. It is frequently present at first as a sense of soreness along the course of the trachea and larger bronchi or in the chest generally. The severe pain often complained of later, especially in the sides of the chest, is due to the straining caused by the cough. Slight pyrexia is often present, especially in children, but is absent in the majority of cases in the adult. The pulse is little if at all quickened in most cases, but it may be greatly so in the very young, and also in the old person. Its rate may be regarded generally as a reliable index of the severity of the attack.

PHYSICAL SIGNS.—The breath sounds are harsh, expiration being prolonged and accompanied by sibilant and sonorous rhonci of quickly varying intensity, particularly after an attack of coughing and expectoration.

Diagnosis.—This is generally easy. The chief difficulty lies in determining if the acute bronchitis be the sole disease or merely one of the manifestations of some primary disease. In some cases of typhoid fever, for instance, acute bronchitis may be so marked as to overshadow the typhoid. The same may be the case in measles, in pneumonias, particularly of the broncho-pneumonic type, and in whooping-cough before the characteristic whoop appears. Acute miliary tuberculosis of the lungs may also show for a time apparently nothing but the symptoms and signs of acute bronchitis. Careful consideration of the history, mode of onset, and local and constitutional signs will serve to clear up all doubt.

Treatment.—Divide the disease into three stages for the purposes of treatment, namely, the pre-secretion stage, the stage when secretion is established, and, lastly, the stage of excessive secretion of sputum.

During the first or catarrhal stage, a diaphoretic combined with a full dose of opium, such as the compound ipecacuanha powder, may arrest, or at least modify, the bronchial attack. It is usual to give 10 to 20 grains of Dover's powder with some hot gruel just before going to bed, and to order the patient to take either a hot bath or hot foot-bath. A saline aperient should be administered in the morning. If this fails, keep the patient in a moist atmosphere at a uniform temperature. Steam inhalations often afford great relief, and a patient hardly able to breathe may in a few moments obtain comparative comfort by inhaling steam. A linseed or mustard and linseed poultice applied to the chest will afford much comfort; in other cases fly blisters are of genuine benefit. A few doses of antimonial wine and a calomel purge are often most efficacious. Towards the end of this stage, and when secretion is rather viscid, much may be done to render it more free. Opium should not be given, but the antimonial wine, ipecacuanha, ammonium carbonate, spirit of chloroform, and many similar remedies are most useful.

During the secretion stage somewhat similar remedies to those just mentioned are helpful, often combining, when necessary, an alcoholic stimulant or general tonic.

Should the sputum become excessive, mineral acids, such as the dilute nitric acid (m 10), with the dilute hydrocyanic acid (m 2 to 3,) and sometimes belladonna, are of value. Sometimes during this stage the sputum becomes not merely too copious, but also foetid, and antiseptics must be used; but this is more likely to occur in chronic cases.

In all the stages of bronchitis there is an element of spasm, and it may be so severe as to demand special

treatment. Nitrites, nitro-glycerine, and even the inhalation of nitrite of amyl are frequently of very great benefit to the patient.

CHRONIC BRONCHITIS.

Etiology.—It is a disease mostly of adult and maturer years, being rare in young people. Men suffer more than women, as is the case in all chronic lung disease. It is most common in winter, particularly in damp, variable climates. The “chronic winter cough” common in many old people, which recurs every year with gradually increasing severity, is a mild form of the disease. Chronic bronchitis may follow upon the acute, but more often arises independently in the course of unhealthy occupations, or in the course of other diseases, *e.g.*, working in dust-laden atmospheres, or during lung, heart or kidney disease. It also occurs in gout.

Morbid Anatomy.—The lumen is generally narrowed in the larger, and dilated in the smaller tubes. The normal epithelium is largely replaced by small oval cells, often imperfectly covering the denuded basement. The other coats are infiltrated with cells, and the muscular and fibrous elements therein may be increased. The mucous glands are usually atrophied. Morbid changes in the lungs and other organs are generally also seen, *viz.*, emphysema, enlargement and pigmentation of the bronchial glands, dilatation of the right side of the heart and venous congestion of the abdominal organs.

Symptoms.—Cough and expectoration are the chief symptoms in all the varieties of chronic bronchitis. In the mild varieties, *e.g.*, winter cough, the cough comes on

with the cold, damp weather and lasts during the winter. It is accompanied by a mucous or muco-purulent sputum easily brought up. In other varieties it is generally more severe and persistent, and more liable to exacerbations. It is usually worst in the morning. The sputum may be scanty, viscid and difficult to bring up ("dry catarrh"), or it may be plentiful, muco-purulent or purulent, and often typically nummulated. The term "bronchorrhœa" or "chronic pituitous catarrh" is used when the sputum is abundant. It is usually watery, frothy and mixed with muco-purulent masses. The term "fœtid or putrid bronchitis" is used when the breath and the sputum are fœtid. Dyspnœa is usually present sooner or later. It is generally due to lung emphysema.

PHYSICAL SIGNS.—Crepitations are common at the bases of the lungs; but otherwise the physical signs are much the same as in acute bronchitis.

Prognosis.—It is a disease which generally gets worse with recurring attacks and advancing years. A change of climate may make the outlook brighter.

Treatment.—For an ordinary case of "winter cough" much may be done by warm clothing, residence in a dry and warm climate during the colder months, and general attention to the health of the patient. Any disease of which the bronchitis is either a clinical feature or a sequel should be treated. Most of the expectorants enumerated under acute bronchitis may be used with benefit: perhaps the most generally useful expectorants are ammonium carbonate (5 grains) and potassium iodide (10 grains) in combination, while terebene, balsam of copaiba, and alkalies are all beneficial. Inhalations of steam impregnated with eucalyptus or terebene are of value where the cough is irritating and expectoration difficult. Locally, counter-irritation should be tried: the compound turpentine liniment,

small fly blisters and croton liniment all being excellent. If the sputum becomes foetid, creasote or carbolic acid may be used for inhalation, and guaiacol (2 parts) and menthol (10 parts) in olive oil (88 parts) given with the intra-tracheal syringe (1 drachm twice or thrice a day). The results of the bronchitis, especially the emphysema, may demand attention, and the compressed air treatment is well worth a trial, while, for the engorged heart and pulmonary circulation, the Nauheim baths and mountain-climbing exercises of Oertel may be recommended in comparatively mild cases. Cardiac tonics are often called for, and in a case of extreme cyanosis bleeding may even be necessary to save life. Oxygen inhalations should not be forgotten where dyspnœa is severe, and the very common spasmodic element of many cases of bronchitis should be met by administering nitrites, nitro-glycerine, and, where very urgent, steam inhalations and hot poultices. Many patients sleep badly, but improve with an occasional sleeping draught, and, in temperate patients, by a small allowance of alcohol in hot water.

PLASTIC OR FIBRINOUS BRONCHITIS.

DEFINITION—A rare form of bronchitis, generally chronic, in which casts of the smaller bronchial tubes are coughed up.

Etiology.—It is twice as common in males. Most cases occur between the ages of fifteen and fifty. Groups of cases may occur, either in several members of the same family or in those living in the same district. Like ordinary chronic bronchitis it is most common in winter, but nothing definite is known as to its causation. An association with

tuberculosis is frequent, and also, though less frequently, with measles, influenza, typhoid, scarlet fever, cardiac disease, rickets, and with certain skin diseases, *e.g.*, pemphigus, herpes, impetigo. Diphtheritic or fibrinous laryngitis sometimes extends to the lungs, forming casts in the bronchi, but this has nothing to do with this disease.

Morbid Anatomy.—The casts are mostly composed of mucin or muco-fibrin. They may be absent altogether after death or present in few or many bronchi, loosely adherent to their walls or lying free. The bronchi themselves may show little or no change, or present the appearances of chronic bronchitis. Emphysema of the lungs is almost constantly present.

Symptoms.—Acute cases are rare. In either case the symptoms resemble those of ordinary bronchitis; but cough and dyspnoea are usually more paroxysmal and more relieved by the bringing up of the sputum. This consists of mucus containing rounded translucent masses which, when unrolled or disentangled under water, are found to be hollow casts of the smaller bronchial tubes. They are often several inches long and may show many ramifications. They have a characteristic stratified appearance, probably due to their being added to after they reach the larger tubes in their upward course. Hæmoptysis is not common but may be profuse. It usually follows, but may precede or accompany, the expectoration of the casts. There may be only one such attack throughout life, but usually there are recurrent attacks at varying intervals of days, weeks, months, or even years. The patient is comparatively well in the intervals between the attacks. The disease may last for years (ten, or even twenty) and generally tends to get worse. The physical signs are much the same as in ordinary bronchitis.

Treatment.—The treatment is most unsatisfactory. The iodide of potash should certainly be tried, and emetics to clear the bronchial tubes may give great relief. Lime water is said to dissolve the casts, and papain has been also commended. Such remedies are usually administered by inhalation as a finely atomised spray.

BRONCHIECTASIS.

DEFINITION—Dilatation of the bronchi secondary to disease of the bronchial walls or surrounding the lung.

Etiology.—Weakening of the bronchial wall must be produced before dilatation follows. This weakening may be brought about either from within or from without. In the former case the bronchial secretions may accumulate from obstruction to their escape and distend the tube, or there may be loss of elasticity in the walls themselves, due to inflammatory changes therein and consequent coughing, as in bronchitis: in the latter case the changes occur first in the surrounding lung and afterwards invade the bronchial walls, as in acute and chronic pneumonias of various kinds. In most cases there is at work the combination of accumulated secretion, damaged bronchial walls, and the pressure effects of coughing.

Morbid Anatomy.—The dilatations show two main forms, viz., cylindrical or fusiform and saccular or globular. The former usually occur in the larger and the latter in the smaller tubes, and both may occur in the same lung. Both lungs are usually affected, though one much more so than the other. A congenital form has been met with in which the disease affects one lung only, though it is universal in that lung. In the usual, *i.e.*, the acquired variety, it is most common in the bases of the lungs, except in cases of

tubercular origin. Occasionally it is present pretty generally throughout one or both lungs. The individual dilations rarely exceed the size of a hen's egg, even in the globular form, in which they are largest. They may be completely or partially filled with a muco-purulent or purulent secretion, which is often foetid. Various cocci may be present, but the factor is due to saprophytic bacteria, and Limmitzer and others have described a special bacillus present in such cases. The inner surface of the cavity is usually smooth and only occasionally ulcerated, but the normal columnar epithelium is replaced by a flattened variety. The wall is usually thinned, sometimes so greatly that the different coats cannot be recognised. Even when the wall is not thinned, the muscular tissue is atrophied and largely replaced by fibrous tissue. The lung tissue around may be normal, particularly in cases which have originated within the bronchi, as in bronchitis or obstruction of the tubes from foreign bodies or other agents. In other cases the lung shows changes of various kinds. The commonest is a chronic interstitial pneumonia with marked contraction of the pulmonary tissue. This is often accompanied by thickened and, but not always, adherent pleura. Emphysema is common. The changes in other organs are dilatation of the right side of the heart and the results of backward pressure. A form of bronchiectasis affecting chiefly the bronchites is met with in acute miliary tuberculosis in children.

Symptoms.—Special symptoms do not arise until the dilatations become considerable. Spasmodic cough, coming on at comparatively rare intervals and accompanied by abundant sputum, is the characteristic symptom. It usually occurs in the morning, but may be induced by a change of posture, probably from the secretion flowing from the dilatation into a normally sensitive tube. The sputum is large in quantity, grey or greenish in colour,

thin and purulent. When allowed to stand it separates into two well-marked layers—an upper, watery one, frothy at the top, with muco-pus suspended in it, and a lower, thick, granular layer consisting of pus, granular detritus and so-called fatty crystals. It sometimes contains also soft greyish-yellow masses, an eighth of an inch or more long, known as “Dittrich’s” or “Traube’s plugs,” consisting of leptothrix filaments, leucocytes, red blood-cells, crystals of hæmatoidin and so-called fatty crystals. Other symptoms may be present. Hæmoptysis is rare. Dyspnœa is rarely seen except on exertion, except when emphysema is present. Abscess of the brain may follow.

Diagnosis.—Paroxysmal cough, accompanied by profuse, often foetid, expectoration, coming on at comparatively rare intervals, indicates bronchiectasis; but in many cases the disease cannot be diagnosed.

Prognosis.—The progress is usually very slow and the patient may appear healthy and live an active life for years. He generally succumbs at last to some complication or intercurrent affection.

Treatment.—The treatment for chronic bronchitis is applicable; but, in addition, the great tendency to decomposition of the contents of the dilated bronchi must be kept in view. One excellent method is to give intra-tracheal injections of one drachm of the following mixture:—menthol ten parts, guaiacol two parts, and sterilised olive oil eighty-eight parts. This may be ordered twice, or even thrice, daily. Solutions of creosote, carbolic acid, terebene and other agents may be atomised and inhaled. Iodoform in solution has been injected into the cavities through the chest wall. Surgical interference has likewise been recommended, but should only be undertaken after careful consideration, as it is by no means free from danger.

Lastly, remember the great risk of septic absorption, and that every attention must be paid to keeping up the patient's strength, with which object cod-liver oil, iron and other tonics should be ordered. Quinine is often of special value where the sputum has become fetid.

ASTHMA.

DEFINITION.—A disease characterised by spasmodic attacks of dyspnoea, during which the respiratory movements are diminished.

Etiology.—Age, sex and heredity all seem to play a part. It usually occurs before ten or between twenty and forty-five. The male sex is much more frequently attacked, and the disease often runs in families, particularly in those of a neurotic temperament. The nervous element plays a most important part in the causation of asthma, for, apart from a nervous diathesis, attacks have been traced to mental conditions of various kinds, such as fright, grief, anger, &c. Further, the nervous stimulus may be of a reflex nature and come from—(1) Any part of the respiratory mucous membrane, either the nose, trachea, or bronchi, following upon the scent of flowers, &c., or the emanations from cats, &c.; or upon irritation from inflammation or nasal polypi, &c.; (2) any other part of the body, such as distended stomach, intestines, disordered genital organs, or even cold suddenly applied to the skin. Hence climate and atmosphere have a great influence in its causation. Going from the town into the country or into a dusty atmosphere may induce an attack. These facts all suggest that the disease is due to disordered innervation producing a spasm of some of the respiratory muscles. A spasm of the diaphragm has been suggested, so has paralysis of the bronchial muscles, but the phenomena of asthma are not explained by either of

these two theories. They are, however, consistent with spasm of the muscular fibres of the smallest bronchi, accompanied by catarrh of their mucosa. The characteristic sputum proves the existence of such a catarrh, and Curschmann, who specially directed attention to it, suggested the name "bronchiolitis" for the disease.

Morbid Anatomy.—There is no evidence of any morbid change other than the catarrhal condition of the small bronchi suggested by the characteristic expectoration which appears usually towards the end of a paroxysm. It consists of small translucent masses or pellets, like sago grains, which often contain spirally twisted bodies (Curschmann's spirals), believed to be casts of the smaller bronchi. They are found also in other lung diseases, *e.g.*, fibrinous bronchitis and capillary pneumonia. The sputum also contains sharp-pointed octahedral crystals (Charcot's crystals) and large numbers of eosinophile leucocytes. These cells are believed by some to be related to the catarrh of the bronchi, and a form of bronchitis affecting the larger tubes has been described under the name "eosinophilous bronchitis," in which the sputum contains, as in asthma, large numbers of eosinophile cells. The circulating blood of the asthmatic patient also contains an excess of eosinophile leucocytes.

Symptoms.—Premonitory symptoms are common. They may appear days or hours before the attack and vary greatly in character. Depression or buoyancy of spirits, irritability, drowsiness, headache, giddiness, neuralgia, dyspepsia, profuse diuresis have all been noticed. The attack usually begins at night. The patient wakes up with a feeling of suffocation. His face is anxious, the eyeballs staring, the nostrils dilated, and the mouth open. Cold perspiration breaks out over the face and neck. The head is thrown backwards or bent forwards, the arms lean upon the

nearest support, the shoulders are raised and the chest fixed in the position of full inspiration. The diaphragm is depressed and moves but little. The patient refrains from speech in the effort to save all his breath. Breathing is noisy and slow, expiration being prolonged. Cough and expectoration come on when the paroxysm is declining. The attack may last only about half-an-hour or be continued with slight intermissions for twenty-four hours or more. The short attacks usually subside suddenly, the long ones gradually. Recurrences may come on at fairly regular or very irregular intervals; but they generally cease altogether at puberty. In later life the disease tends to get worse as time goes on, though death rarely occurs during an attack.

Diagnosis.—Paroxysmal dyspnoea comes on in other diseases than in asthma, *e.g.*, in laryngeal disease, in cardiac disease, and in thoracic aneurism. The last-mentioned is most likely to be confounded with asthma, from which it is to be distinguished by the greater severity and duration of the attacks and by not yielding to the ordinary remedies for asthma, as well as by the history and some or other of the positive signs of aneurism.

Treatment.—For the relief of the attack try nitrite of amyl in 3 minim capsules. One of these broken in the handkerchief and inhaled often altogether arrests or greatly lessens the spasm. Other antispasmodics are stramonium, lobelia and nitre paper, the fumes of which are of much value. Many asthma cures, whether powders or cigarettes, contain these or similar ingredients. To a non-smoking patient the slight nausea produced by smoking tobacco is beneficial. Internally, nitroglycerine ($\frac{1}{100}$ to $\frac{1}{50}$ of a grain), the tincture of belladonna (5 to 10 minims) and the ethereal tincture of lobelia (10 to 15 minims) are serviceable. Opium gives great relief but is apt to induce the habit of taking the drug with dangerous frequency, and

chloroform anæsthesia is also of value, but should only be administered by the medical attendant. An emetic for an overloaded stomach or a brisk purge may give prompt relief. Between the attacks try the use of the nitrite of sodium in $\frac{1}{2}$ to 2 grain doses thrice daily, or the iodide of potash in 10 grain doses: we may combine with one or other of these remedies arsenic in small doses. Be careful to examine for and to treat any morbid condition in the upper air passages. Dress the patient warmly, choose, if possible, a climate and habitat where he is nearly immune from attacks, and pay attention to diet and to any constitutional element, such as gout, which may be present in his case. Try also, by pulmonary gymnastics and sometimes by the use of the compressed-air chamber, to prevent an increase of the emphysema invariably present.

OBSTRUCTION OF THE TRACHEA AND BRONCHI.

This subject requires only a word or two. There are three sources of obstruction—viz., entrance of a foreign body, stricture, and compression from without. A foreign body rarely lodges in the trachea. If it does it will cause sudden death if it completely obstructs it, and spasmodic dyspnœa if the obstruction be partial. It much more commonly lodges in one or other bronchus, usually the right, and may cause ulceration, hæmorrhage, abscess or septic pneumonia. Stricture is most commonly due to syphilis, though it may occur in tubercle or leprosy. The lower part of the trachea and upper parts of the bronchi are most usually affected. Obstruction of a bronchus causes dyspnœa and stridor with enfeebled respiratory sounds on the affected side, though the percussion note remains

normal. Compression of the trachea or bronchi from without may be caused by goitres, aneurisms, mediastinal tumors, or diseased bronchial glands. The symptoms resemble those of stricture.

Treatment.—Always think of a possible syphilitic element in the case and try the iodide of potash with mercury. Sometimes a stenosed trachea can be treated by dilating the stricture, while tracheotomy is necessary where life is in danger. Antispasmodics give much relief in many cases, and may be the only method of treatment possible. A foreign body, if present, will have to be removed by surgical measures.

IV.—DISEASES OF THE LUNG.

Certain abnormalities arise in the lung from obstruction of either the air or the blood supply. The former gives rise to emphysema and collapse, the latter to venous congestion, œdema and hæmorrhage.

EMPHYSEMA.

DEFINITION.—An over-distension of the alveoli and rupture of septa in the lung.

Etiology.—Frequently recurring violent cough seems to be the most frequent cause, and hence it is common in those suffering from chronic bronchitis. Similarly, any violent expiratory effort with a closed glottis and compressed chest walls conduces to its production—*e.g.*, the playing of the cornet and other wind instruments. Obstruction to the nasal passages is said to act in the same way. It is more easily produced in some people than in others, and it is therefore thought that a certain malnutrition or weakness

of the pulmonary tissue, either hereditary or acquired, must pre-exist.

Morbid Anatomy.—The lungs are both, though unequally, affected. They are more voluminous and do not collapse when the chest is opened. They show a number of bullæ all over the surface, but particularly at the apices, anterior margins, roots and bases, where the bullous projections may be as large as a hen's egg. The tongue-like projection of the left lung, which lies on the heart, may be so enlarged that it entirely covers that organ, causing the usual area of superficial cardiac dulness to disappear. These bullæ are much lighter in colour than normal, and their contained air can be squeezed from them into adjacent parts. Crepitation is diminished and the lung substance feels softer. It has been likened to the sensation got by pressing a silk bag full of feathers. Microscopically, the air vesicles are seen to be much enlarged, their septa thinned (particularly the elastic tissue, which is probably the first constituent to suffer) and broken. The epithelial cells lining the air vesicles play a passive part, and are usually unaltered. This form of emphysema is usually called vesicular, to distinguish it from another form of occasional and much rarer occurrence, known as interstitial, in which the air collects as small and separate globules situated in the interalveolar or interlobular tissue. This form may follow upon the vesicular, particularly when there is very violent cough, or in toxic respiratory disease, such as diphtheria. It may follow, also, upon wounds of the chest wall. It is local in its distribution. It also is to be noted that a local form of vesicular emphysema may occur as well as the general form above described. It occurs when a swelling of the bronchial mucous membrane, as in bronchitis, allows of free entrance of air in inspiration and its diminished expulsion in expiration, or when consolidation of one part leads to an excess of air, during inspiration, entering contiguous parts, as in tubercu-

lar and other forms of lobular consolidation. It may be acute or chronic. The former usually disappears with the removal of the cause, but the latter, which usually follows upon repeated attacks of the acute, or occurs independently in those with weakened pulmonary tissue, as in wasting diseases and senility, is permanent and forms bullæ like those mentioned above. They are most frequent along the anterior and inferior borders of the lower lobes and tongue-like projection of the anterior border of the left lung, which overlies the heart, but may be seen in the upper parts and elsewhere. This form has been called also substantial, compensatory or senile emphysema. Associated with these changes in the lung parenchyma there are catarrhal changes in the bronchi (many of the smaller ones being dilated) and hypertrophy and dilatation of the right side of the heart, accompanied later, it may be, by general systemic venous congestion and dropsy.

Symptoms.—The chief symptoms are dyspnœa, cough, cyanosis, and severe emaciation. They are more or less dependent upon the heart. When cardiac compensation is complete, there may be no symptoms at all, even in well-marked cases of the disease. The dyspnœa, when present, varies in severity. It may be intermittent or persistent, and is usually aggravated by exertion or bronchitis. The cough depends on the attacks of bronchitis, and hence is worst in winter. It becomes more persistent as time goes on, and is attended by a scanty frothy expectoration. The cyanosis is often extreme. It is greatest after a fit of coughing, when the face may become blue or even purple without the patient showing much general distress. The emaciation comes on later, and is largely due to disturbed digestion.

PHYSICAL SIGNS.—The chest becomes rounded in shape, barrel-shaped as it is called, owing to the rounding of the shoulders and the forward projection of the sternum and

costal cartilages. The intercostal spaces are widened, the clavicles prominent, and the thorax elevated, giving a shorter appearance to the neck. The respiratory movements, though apparently forcible, are restricted, inspiration being short and expiration prolonged. The apex beat is masked, but there is usually well-marked epigastric pulsation. These appearances form a striking and easily recognised picture in well-marked cases. Percussion gives a full drum-like note, often spoken of as hyperresonant; it may be actually tympanitic. The cardiac, hepatic and splenic dulness is diminished owing to the overlapping borders of the lung. On auscultation, inspiration is short and feeble, expiration is greatly prolonged and often harsh and accompanied by rales or sibilant ronchi. Vocal resonance is diminished. The heart sounds are normal, though the pulmonary second sound is usually accentuated. In later stages there may be a tricuspid regurgitant murmur.

Prognosis.—The disease is usually slow but progressive. It is rarely fatal in itself, and patients may live to a good age. The progress will depend upon the frequency and severity of the bronchitic attacks, the state of cardiac compensation, the intercurrent of pneumonia or renal disease.

Treatment.—Most patients who suffer from emphysema suffer also from bronchitis, and it is generally requisite to adopt the treatment for the latter condition when it develops. On the other hand, certain of our patients may suffer from asthma, and to them the treatment for asthma is applicable.

Once a patient has developed emphysema it is difficult to cure it, although much may be done to render life more endurable and to obviate an increase of the condition by preventing fresh attacks of bronchitis or asthma. Residence at a high altitude is sometimes beneficial, although it is often better to advise the patient to spend the colder months of

the year in a warm, equable climate, such as Egypt. The use of inhalations of condensed air appears to afford a certain amount of benefit. Remember the strain on the right heart, for the relief of which venesection may even be necessary, while oxygen inhalations and sometimes cardiac tonics are helpful. Iodide of potash has been recommended, and it is certainly of much service in chronic bronchitis; but it is often of more importance to study the general health of the patient, giving cod-liver oil, iron and strychnin where desirable, and to see that our patients are warmly clothed and have work suitable for the pulmonary condition.

COLLAPSE—ATELECTASIS.

DEFINITION.—An airless condition of lung alveoli, caused by either—
(1) their failure to expand at birth (the congenital form), or
(2) the loss of their air in later life (the acquired form).

THE CONGENITAL FORM.

Causes.—Blocking of the bronchial tubes by mucus or meconium or other foreign matter is usually held to be the most frequent cause, but it may follow obstruction to the bronchi by other causes, such as compression from without of enlarged glands. Pressure upon the thorax or upon the diaphragm may cause it. Feeble respiratory power may also produce it, particularly in premature children.

Morbid Anatomy.—It generally affects small areas, each comprising only a few lobules, in the bases of the lung, particularly posteriorly, but it may affect considerable areas, even a whole lobe. The collapsed parts are dark red, firm, non-crepitant and sink in water. Such areas are

often capable of expansion, as can be proved after death by means of a blow-pipe, and they often do expand in life after a few days, but when the condition has continued for more than a few days inflation becomes increasingly difficult, and then impossible, owing to a loss of the alveolar epithelium and fibrous adhesion between the alveolar walls. Later, considerable atrophy may occur and the collapsed area may be replaced by a diminutive scar.

Symptoms.—The child may die at birth, but if it lives it does so usually for only a few days. Pallor, combined with cyanosis, tending to get deeper, is prominent. Breathing is shallow, and there may be dyspnoea and slight convulsions. The child is sleepy and feeble, with a feeble cry and little or no power to suck. Slight cases recover.

Treatment.—Stimulate the child by dipping him alternately into hot and cold baths. Occasionally artificial respiration is successful, but it must be admitted that a fatal result is practically certain in severe cases.

THE ACQUIRED FORM.

Causes.—The two chief ways in which collapse of a normally expanded lung is produced are—(1) By interfering with the entrance of air into the lungs; (2) by driving it out after it has entered. The first is seen in all forms of obstruction of the bronchi, *e.g.*, inflammatory swellings of the mucous membrane (best seen in the smaller bronchi), the presence of secretions, new growths or foreign bodies in the lumen of the tubes, the obliteration of the tubes by pressure from without, as in tumors, aneurisms, &c. Little or no air enters with each inspiration, and, whether air from the lung leaves with each expiration or not, it is gradually absorbed until the part becomes airless. The

second method is seen in the accumulation of air or fluid in the pleural cavity pressing the lung from without. The same thing may happen by the pressure of a tumor or a pericardial effusion or abdominal distension. In addition to these two a third must be added, viz., deficient power of expansion of the lungs. It is seen in rickets in children, where there is a lack of rigidity in the chest wall, or in old age, from muscular weakness, and, in short, in any case, *c.g.*, fevers, where there is marked weakness in the movements of the thorax.

Morbid Anatomy.—When caused by outside pressure, such as pleural effusion, the collapse may be total, when the whole lung is pushed backwards, inwards and upwards towards the root, forming a dense dark non-crepitant tissue which sinks in water. In other cases it is local, affecting single or scattered patches, particularly in the bases and free borders, forming bluish-red, depressed, firm, airless areas. The surrounding parts of the lung may be emphysematous, or congested and œdematous. The collapsed areas are intensely congested and capable, for a time, of natural or artificial expansion; but after a time they atrophy and form fibrous scars.

Symptoms.—They may be lost in those of the causal condition. There are *none* if the areas be small, but when large, as in whooping-cough, there may be much dyspnoea, with cyanosis, dulness on percussion, and diminution of the breath sounds on auscultation.

Treatment.—Try at once to remove the obstruction if one is present and to permit the free entrance of air into the lungs: thus, in diphtheria, give emetics, and, where necessary, resort to intubation or tracheotomy. In other cases, where there is no such obstruction in the bronchial tubes, stimulate the patient, sometimes by cold douching

associated with hot baths, sometimes by the exhibition of carbonate of ammonia, spirits of chloroform and other stimulating remedies; while the patient's strength is kept up by the frequent administration of easily digested and nourishing soups, &c. Sometimes pleural effusions require tapping.

SUB-GROUP (a)—*INTERFERENCES WITH THE CIRCULATION IN THE LUNGS.*

Morbid conditions of the lungs arise through interference with the circulation of blood, as well as with that of the air. They are—passive congestion, œdema and hæmorrhage.

PASSIVE CONGESTION.

There are two forms of passive congestion—the ordinary mechanical and the hypostatic.

MECHANICAL VENOUS CONGESTION.

Mechanical venous congestion is caused by anything which interferes with the return of the blood from the lungs to the left side of the heart, *e.g.*, mitral stenosis or other valvular lesion, or pressure on the root of the lung by a tumor or aneurism.

Morbid Anatomy.—The lungs are swollen, except in very old-standing cases, in which they may be much about the normal size. The lung substance is more resistant and difficult to cut, and the fresh section shows a brown

colour and dry surface, hence the term brown induration. Microscopically, the alveolar capillaries are swollen and beaded, the septa thickened and infiltrated with cells containing pigment, and the alveolar cavities show a number of desquamated epithelial cells which contain variously altered red blood-cells and blood-pigment.

Symptoms.—In very slowly produced cases, *e.g.*, those due to a lesion of the left side of the heart, in which compensation is complete, there are no symptoms, but when compensation fails there are cough, dyspnoea, and expectoration. The sputa may be hæmorrhagic, but they always contain the pigmented epithelial cells.

Treatment.—Mechanical venous congestion should be treated by direct or indirect relief. Sometimes we can remove from 20 to 30 ounces of blood and thus afford direct relief, or we can give diuretics and purgatives and so draw off indirectly the fluid from the blood-stream. Tapping dropsical effusions, where these are present, should certainly be attempted in severe cases.

HYPOSTATIC CONGESTION.

This condition is brought about by two factors—cardiac weakness and gravity. It may occur, therefore, in all cases where the heart's action becomes weakened for a time, as in prolonged fevers and other weakening diseases, particularly if the patient lies in bed in one position for a long time. The hypostatically congested lung is particularly susceptible to attack by germs, hence the congestion is often followed by pneumonia.

Morbid Anatomy.—The bases and posterior parts of the lung are the parts chiefly affected. They are more bulky and congested than normal. In severe cases they

feel almost solid. Microscopically, the same changes are seen as in the mechanical form, but the alveoli contain far more cells, many of which may be leucocytes.

Symptoms.—They are much the same as those of the mechanical form, but dulness and crepitations may be detected as well.

Treatment.—Hypostatic congestion is often the sign of approaching death and of the necessity for vigorous stimulation, but in a mild degree it is of less grave importance, although its presence should make the physician consider the desirability of frequently altering the patient's position in bed and should induce him to use alcoholic and other diffusible stimulants.

ŒDEMA.

Causes.—In intense or prolonged congestion the blood-serum transudes in excessive quantity into the alveolar septa and into the alveoli. It also occurs in inflammations of the lung parenchyma. The altered nutrition of the capillary walls brought about by either of these causes makes them more permeable. Œdema occurs very frequently towards the end of many fatal illnesses, in many cases during the death agony, particularly in chronic cardiac and chronic Bright's disease, in cachexias and certain forms of cerebral disease.

Morbid Anatomy.—The whole lung may be affected, but the condition is commonest at the bases. The affected parts are bulkier and heavier than normal, pit on pressure, and the section is watery-looking, a blood-stained watery

fluid exuding spontaneously or on pressure. Microscopically, there is little change to be seen in most cases, beyond sometimes a little swelling of the epithelium of the alveoli.

Symptoms.—They are usually masked by those due to the primary disease, but they may become marked when the œdema is of rapid onset and advance, as in some cases of Bright's and of cardiac disease. They are dyspnœa, often cough, increasing dulness, with crepitations at the bases of the lungs.

Treatment.—Where œdema is associated with backward pressure, try to relieve it by diuretics, purgatives, by tapping any dropsical effusions in the pleural sacs, and in very bad cases by venesection. Give stimulants, and especially the aromatic spirit of ammonia and the spirits of ether and chloroform in drachm doses (20 minims of each). In children an emetic is sometimes of value, and in practically all cases vigorous local counter-irritation is worth a trial.

HÆMORRHAGE.

Hæmorrhage into the lung substance or into the bronchi may escape from the mouth, or infiltrate the lung substance. The escape or spitting of blood is called hæmoptysis. There are many causes of pulmonary hæmorrhage, and hence of hæmoptysis, which may be grouped under the headings of diseases of the lung or air passages, diseases of the heart, aneurism and rupture of intra-thoracic or intra-pulmonary blood-vessels, violent bodily exercise, vicarious menstruation, certain constitutional states, *e.g.*, fevers, purpura hæmorrhagica, &c. . Among diseases of the lung

or air passages the most frequent is tuberculosis. The hæmoptysis of tubercle occurs particularly in the early and late stages of the disease, although it may occur at any time. The early hæmoptysis is often scanty and may be due to diapedesis following upon the tubercular growth around the blood-vessels weakening their walls and rendering them more permeable. When it is considerable, say two or more ounces, it is probably due to ulceration of the vessel walls. The late hæmoptysis is produced in the same way, and by rupture of intra-pulmonary aneurisms, but it is much more frequently copious, and may even amount to a pint or more. It is, however, rarely the immediate cause of death. Of other diseases of the lung which may cause hæmoptysis, cancer, gangrene, and abscess must be mentioned. It may be of considerable amount in these diseases. In lobar pneumonia, on the other hand, it is generally scanty.

Among diseases of the heart liable to cause hæmoptysis, mitral stenosis must be put first. Aneurisms of the vessels within the chest cause hæmoptysis by rupture into the lung substance or into the air passages, when the amount may be so great as to cause immediate death, or it may be small and recurrent for some time before the fatal hæmorrhage occurs. The influence of violent exercise is sometimes seen in heavy weight lifting, in loud singing, in rapid ascents of high mountains. Diminished atmospheric pressure comes also into play in the last instance. In addition to all these diseased and other conditions, hæmoptysis may occur in young people particularly who are apparently perfectly healthy. Cases are on record of one or more suddenly occurring hæmoptyses in persons who show no sign of ill health, either at the time or for years afterwards. Such cases are open to the suspicion of early tubercle, which becomes spontaneously cured, but it is at least doubtful if they are all of this nature, and their causation remains obscure. The expectorated blood is

bright red and frothy, from admixture with air and mucus, and has an alkaline reaction. It is generally attended by cough and a warm saltish taste in the mouth. When great in amount, as in the rupture of an aneurism, it may be pure blood, and when small in amount it may merely impart a blood-streaked or tinged appearance to the sputa. The blood may be swallowed and cause hæmatemesis. In the great majority of cases of hæmoptysis, whatever the cause, the hæmorrhage ceases spontaneously. When the hæmorrhage is into a bronchus, it is probable that in most cases all of the blood escapes from the mouth. A part may at times be aspirated backwards into smaller tubes and into the lung alveoli. When the hæmorrhage is into the lung substance it may fill up the alveoli in its neighbourhood more or less extensively. When the escape is slow, as in diapedesis, the blood will gradually expel all the air from the alveoli, and after coagulation form a solid area, resembling fresh blood clot, within the lung. This is most characteristically seen in the *pulmonary infarct* or *apoplexy* whose causation is twofold—(1) an embolus, blocking a branch of the pulmonary artery, (2) obstruction to the flow of blood through the pulmonary veins. This apoplexy is most commonly met with in the periphery of the lung as a wedge-shaped area of small or large size with the base at the pleural surface. This pleural surface is smooth and unaltered when the apoplexy is recent, but generally shows inflammation when it is older. The apoplexy itself forms a well-defined, solid, airless mass of a dark red colour, like blood clot when recent, but paler when older. A zone of fibrous tissue, due, as in other infarcts, to reactive inflammation, gradually walls it in. It undergoes gradual absorption and forms an indurated pigmented patch. Microscopically, it shows, when fresh, the alveoli filled with blood clot and the alveolar walls intact (very rarely broken), later the blood clot breaks down, the leucocytes increase and become pigment laden. Absorption

may be complete, and in cases where the hæmorrhage was not severe enough to cause death of the alveolar walls, the respiratory function of the part may be restored. In other cases the absorption extends to the dead lung tissue, and the whole patch becomes replaced by newly-formed fibrous tissue. When pyogenic cocci are present in the embolus, or enter the infarction in sufficient numbers, an abscess is formed. Micro-organisms in varying numbers enter most lung infarcts with the inspired air, and probably have some influence in the formation and in the size of the infarct. Multiple emboli produce an infarct more readily than a single one, which may exist without a subsequent infarct. The diagnosis of pulmonary apoplexy is difficult during life. Dyspnœa, cough and hæmoptysis in the course of a mitral case may suggest it, but are equally indicative of pulmonary congestion without apoplexy. If the apoplexies are large, they may produce dull areas, but they are difficult of detection, as they occur most frequently towards the end of the case, often just before death.

Treatment.—When a hæmorrhage is due to pulmonary tuberculosis, place the patient in bed, keep him absolutely quiet and apply an ice-bag over the chest, provided you can ascertain the part of the lung from which the blood is being effused. He may lie on his back or, preferably, on the affected side, so as to keep the other lung clear of blood. Allay fear by giving morphia hypodermically, or a mixture containing liquor morphinæ hydrochloratis (10 minims) and acidum sulphurici diluti (10 to 15 minims), every two or three hours, by the mouth. In a very severe case, and where the continuance of the hæmorrhage is threatening the patient's life, the removal of 10 to 15 ounces of blood from a vein may cause arrest of the hæmorrhage, and it should not be forgotten that if the patient faints the bleeding may cease. Ergotin is of doubtful benefit, but

adrenalin (10 to 15 minims), calcium chloride (10 to 20 grains) and turpentine or terebene are useful hæmostatics. Later, saline purgatives are advantageous because they tend to prevent a recurrence of the hæmorrhage. The diet should be light and administered in small quantities.

Hæmorrhage from a malignant tumor of the lung is generally not very excessive, but cannot be well controlled. Hæmorrhage from the rupture of an aneurism is usually early fatal. Hæmorrhagic infarction is often really beneficial, because, should the patient spit up a considerable quantity of blood, the backward pressure so frequently associated with the condition is thereby greatly relieved.

SUB-GROUP (β)—*INFLAMMATIONS OF THE LUNG.*

Inflammations of the lung present very varied morbid appearances. They may be classified according to their causation or their histological characters. Both these classifications have advantages. The latter is the simpler for it has only to deal with the main morbid histological changes, thus we speak of a fibrinous pneumonia when the air vesicles are filled with an exudate rich in fibrin, of a catarrhal pneumonia when the exudate consists of desquamated endothelial cells, of a suppurative pneumonia when it is purulent, and of a fibrous pneumonia when there is a great increase of fibrous tissue. It has, however, the demerit of raising into undue prominence the morbid changes in the lungs, and of losing sight of the causation of the disease and its real nature. The pulmonary lesions may be the chief morbid changes produced in any of these pneumonias and yet form by no means the most significant part of the disease. In acute cases they con-

stitute *per se* a less important part than the general effect of the disease upon the body as a whole. That is to say, that in so far as they interfere with the respiratory function of a part, sometimes of a very large part, of the lung they do not thereby cause death, or even gravely threaten life, through asphyxia. It is the poison, of which they are the chief sites of generation, which, on its absorption into the system, causes the gravest disease by its hurtful action on all the bodily centres of activity. An etiological classification of lung diseases is therefore the best if it be practicable. It is not enough to take into account the path of entrance only of the causative agent. It is obvious that there are only four ways in which it may attack the lung—(1) By the bronchi, *e.g.*, along with the inspired air, giving what may be called “bronchogenic pneumonia”; (2) by the blood, giving “hæmatogenic pneumonia”; (3) by the lymphatics, as from the pleura, giving “pleurogenic pneumonia”; and (4) by direct extension from a neighbouring organ, when it is usually called secondary. This classification has few, if any, advantages over the anatomic one, as it does not take into account the nature of the causative agent itself. This is generally, if not indeed always, an infective bacterium. Some of these are capable of causing inflammation in the lung just as they are capable of causing it in other organs or tissues. Any one of the general group of the bacteria which cause inflammation or suppuration, *e.g.*, the staphylococci, the streptococcus, Frænkel’s diplococcus lanceolatus, Friedländer’s pneumobacillus, or the bacillus coli communis may cause a pneumonia extending over a large or small part of one or both lungs; but inasmuch as they are in a manner interchangeable, *i.e.*, it is sometimes the one and sometimes the other which causes lesions apparently identical in nature, they may be called non-specific organisms, and hence the inflammations of the lung produced by them, non-specific pneumonias. On the other

hand, the specific organisms, *i.e.*, those which are alone capable of causing certain diseases—*e.g.*, tubercle, typhoid, influenza—may attack the lung and cause specific pneumonias, differing greatly it may be, or but slightly, in their topographical and histological characters from those caused by some of the non-specific organisms. There are certain predisposing causes of great influence in the causation of pneumonia. Cold, injury, fatigue, alcoholism or any agent capable of lessening the resisting powers of the system, such as any acute infection like measles, or any wasting disease like diabetes mellitus or chronic Bright's disease, may enable invading bacteria, powerless to attack a healthy lung, to flourish and cause a pneumonia. These two great divisions—non-specific and specific pneumonias—may be either acute or chronic.

ACUTE NON-SPECIFIC PNEUMONIAS.

The most characteristic and by far the most frequent are those caused by the *diplococcus lanceolatus* of Fränkel. Of these there are two varieties—the lobar and lobular pneumonias, the former of which is the more important.

LOBAR PNEUMONIA.

The lesions it produces in the lungs and the clinical course of the disease are generally so typical and characteristic that the disease caused by it practically constitutes a clinical entity. It is called by various synonyms, the most common of which are simply *pneumonia*, *lobar pneumonia*, because it generally extends over a large part of the lung (a whole lobe or more), *fibrinous* or *croupous pneumonia*, because of the abundant fibrinous exudate

which comes from the blood-vessels and fills the lung alveoli, also *pneumonitis* or *lung fever*.

The bacillus lanceolatus.—This organism is also called Fränkel's diplococcus or pneumococcus because it frequently forms a pair of bacteria so short as almost to appear to be cocci. They soon grow out into short bacilli of a distinct lance-like shape, with their broad bases next each other and their pointed ends away from each other. Each coccus (when single) or each pair is surrounded by a distinct capsule. This capsule is not seen in most artificial cultures, only in the natural growth within the body. This germ is frequently present as a saprophyte in the mucus of the throat or larynx of healthy people. This suggests its reaching the lung substance by the bronchi.

Morbid Anatomy.—As already said, it generally attacks a large area of lung substance, a part of a lobe, a whole lobe (the lower most frequently), or even a whole lung or both lungs. The appearances presented by the affected lung will vary with the duration of the disease. At first there is only congestion of the alveolar capillaries with, it may be, some serous exudate into the alveoli; but it is soon followed by a fibrinous exudate which drives the air out of the alveoli, and, taking its place, converts the lungs into a solid structure, something like the liver to the touch, hence it is called "hepatisation" of the lung. It sinks in water. The hepatised lung is larger than normal. It generally shows some fibrinous exudate on the pleural surface. It cuts easily, the cut section being at first almost smooth and red or mottled red in colour—the stage of *red hepatisation*; later markedly granular and of a mottled grey colour—the stage of *grey hepatisation*. Microscopically, in the former stage, the alveoli are filled with plugs of fibrinous exudate which have the appearance of a network of fibrin threads, in the meshes of which are red and white

blood-cells with a few catarrhal cells, and the interalveolar capillaries are dilated; while in the stage of grey hepatisation the plugs have shrunk a little from the alveolar walls and become more opaque, the leucocytes and catarrhal cells being increased in number and fatty, the red cells somewhat broken down, and the interalveolar capillaries no longer dilated. In favourable cases further degeneration and absorption of the alveolar plugs takes place—the stage of *resolution*—until they become sufficiently softened to be coughed up or absorbed. The air again enters the alveoli, which become provided with a regenerated endothelium, and recovery is complete.

The diplococcus is found in all parts of the exudate in the alveoli throughout the disease, but in greatest numbers where the inflammation is most recent, hence in the spreading margins, *i.e.*, in the inflamed œdematous parts not yet consolidated. It is present in the sputum throughout the whole course of the disease. In the great majority of cases it is restricted to the lung, the pleural exudate, and the sputum; but in a certain number it extends further, either by contiguity to neighbouring tissues, *e.g.*, the pericardium, the mediastinal glands, or to distant organs by absorption into the lymph or blood, and there may set up a similar fibrinous inflammation or suppuration. Thus peritonitis, meningitis, endocarditis (often malignant), arthritis, otitis media, inflammations or suppurations of the subcutaneous tissues, &c., may arise, either during the course of the pneumonia as complications, or after its subsidence as sequels thereof. It must, however, be particularly noted that any of these lesions may occur as a primary disease without any appearance of pneumonia. The following statistics of Netter show the relative frequency of the primary infections by the diplococcus lanceolatus in man—(1) In adults, lobar pneumonia 65·95 per cent., bronchopneumonia and capillary bronchitis 15·85, meningitis 13, empyema 8·53, otitis 2·44, endocarditis and liver abscess

each 1·22. (2) In children forty-six cases were investigated and in twenty-nine it was otitis media, in twelve bronchopneumonia, in two meningitis, in one lobar pneumonia, in one pleurisy, and in one pericarditis. Inflammatory or suppurative changes may therefore arise in any tissue in which the organism finds a lodgment. It has already been stated that it is a usual inhabitant in the throat and nose, and Netter thinks it may pass directly from the nose to the ear. It certainly passes directly from the throat and respiratory passages to the lungs, but it travels to distant organs by the blood-stream, and it can be found in many cases of pneumonia (particularly the severe cases) within the blood, though in small numbers, if a sufficient quantity (about 2 cmm.) be examined. More serious changes still are those produced by the action of the toxin generated by the germ. This is absorbed into the blood and acts upon all the organs of the body—upon the heat centres, causing fever; upon the heart, causing cardiac weakness and failure and leading to the formation of intra-cardiac thrombi; upon the spleen, causing congestion; upon the liver, causing granular degeneration; upon the kidneys, causing congestion and similar degeneration or even nephritis and leading to alterations in the urine, *e.g.*, albuminuria and diminished excretion of chlorides; upon the bone marrow, leading to an excessive formation of polymorpho-nuclear leucocytes and a pronounced leucocytosis (sometimes 50,000 to 60,000 per cmm.) in the circulating blood; upon the nerve centres generally, leading to great depression.

Symptoms.—A typical case in an adult mostly comes on suddenly with a severe chill or a rigor. Some malaise of a few days' duration occasionally precedes it. It is followed by fever, the temperature usually rising rapidly. It may be even 104°F. or 105°F. shortly after the onset of the illness. It continues high, with only slight remissions,

during the course of the illness, which usually lasts acutely for about seven days. Pain in the side is another early and frequent symptom. It is usually very severe and is aggravated by deep breathing and by coughing. It is due to the pleurisy, and usually subsides after a few days. Breathing becomes more rapid, the respirations reaching 40 or 60 or even more per minute. This rapid shallow breathing is often very striking, necessitating the use of some of the accessory muscles of respiration, hence the *alae nasi* are often seen to be working. There is, however, no difficulty in breathing, only rapidity because of the shallowness, due partly to the pain and partly to the diminished respiratory surface in the lung. The pulse is also quickened, but not usually so markedly as the respirations. Hence the pulse-respiration ratio, which in health is 4 to 1, is altered, becoming 3 to 1 or 2 to 1, or even less. Cough of a short, hacking character comes on early. It is frequent and restrained because of the pain in the side. Though dry to begin with, a sputum soon appears, at first frothy and mucoid but soon becoming viscous, tenacious and *blood-stained*, hence it is commonly called *rusty*. Owing to its viscosity and tenacity it sticks to the teeth and lips and is difficult to expectorate. These characters are not met with in any other disease, though they may be present when this disease occurs as a complication of another—*e.g.*, influenza, tubercle. There is thirst and loss of appetite as in other fevers. The urine is decreased in amount, its urea increased and chlorides diminished. Albuminuria is frequent. The face is usually flushed, particularly on the side of the pneumonia, and the skin hot and dry. Herpes of the lips or *ala nasi* is common after the fourth day, and usually appears in successive crops. Headache and mental wandering, or even severe delirium, occurs. There is often great general prostration.

THE CRISIS.—This occurs on an average on the seventh day, but may be earlier or much later. The temperature

falls to or below the normal within six to twenty-four hours : it is often ushered in by a profuse sweating or other critical evacuation. The patient feels comparatively comfortable and convalescence begins.

PHYSICAL SIGNS.—Fine crepitations and quieter, weaker or harsher breath sounds over the affected lung are the earliest signs. Later, when the stage of consolidation is reached, there are the further signs of diminished movement (recognised by both inspection and palpation), of increased vocal fremitus, of dulness and increased resistance on percussion, of bronchial breathing, and of increased vocal resonance (bronchophony). Neither the dulness nor the resistance are so great as in pleural effusion, where the note has a wooden sound. Bronchophony is absent if the bronchi do not remain patent but become filled with exudation (massive pneumonia). When resolution sets in, coarse crepitations appear and the dulness gradually disappears (seven to fourteen days). Departures from this typical history are often met with, particularly in children and old people. In the former the illness may begin with *vomiting*, *convulsions*, delirium, pain in abdomen, and severe headache and strabismus, thus simulating tuberculous meningitis. The temperature may rise more gradually, but usually reaches a high degree early. There may be no sputum, and the pulse may be very quick (160 to 180) without indicating any special danger, whereas in an adult it would be grave, as indicating severe prostration. In old people the onset is insidious. It may also be ushered in with vomiting. There is often neither cough nor sputum and the physical signs are often unsatisfactory. The fever is rarely high, seldom reaching even 102°F., particularly in drunkards. The pulse is feeble and irregular, and physical signs may be slight. Delirium, if present, may be of a low muttering type, attended by marked prostration. Coma may supervene. Delirium is always a serious symptom, whether it appears in children, adults or old people.

Its intensity may be taken as an indication of the severity of the toxæmia. It may simulate acute mania in adults or delirium tremens in drunkards.

Complications.—From what has already been said of the extent to which the pneumococcus may be distributed throughout the body, it is clear that inflammatory or suppurative affections of any part of the body may occur, but experience has shown that certain affections are more common than others. *Pleurisy* is so constant that it constitutes rather a feature than a complication, but it does not usually cause attention to be drawn to it other than by the pain in the side. It may be regarded as a complication when it gives rise to considerable effusion, whether serous or purulent (*empyema*), particularly when this occurs on the opposite side to the pneumonia. When irregular fever returns after the crisis and dulness remains or increases, an exploring needle should be used. *Pericarditis* is much less common than pleurisy. It is more frequent and less serious (though always grave) in children than in adults. *Acute Endocarditis.*—This is more common than pericarditis. It is most common where pre-existing chronic valvular disease exists. The simple and malignant varieties appear to occur in almost equal numbers. *Jaundice*, which is usually slight, is not uncommon. It does not appear to be of any special significance. *Meningitis* may come on early or late in the disease. It is a grave complication. *Peripheral neuritis, acute arthritis, acute nephritis, and parotitis* are all rarely met with complications.

Diagnosis.—In typical cases it is easy enough, but in deep-seated pneumonias, and in those of children, old people and drunkards, it is often difficult. In children it may be mistaken for meningitis or cerebral abscess, but the headache and delirium are often not so lasting as in the latter diseases. When vomiting and abdominal pain

are severe it may simulate appendicitis, peritonitis or intestinal obstruction. The continued fever, the altered pulse-respiration ratio and the physical signs are the best guides in all cases. Microscopic examination of the sputum for the pneumococcus is also of value.

Prognosis.—It is gravest in infancy and old age. The mortality is least between five and fifteen years of age, being a little over 3 per cent. It steadily increases after twenty years of age, being about 22 per cent. in the third decade, 30·8 per cent. in the fourth, 47 per cent. in the fifth, 51 per cent. in the sixth, 65 per cent. in the seventh. Apart from age it is greatest in those debilitated by intemperance, privation or disease. Though death is rarely, if ever, due to asphyxia, but mostly to cardiac failure, and more rarely to the general prostration or the high temperature, yet the danger increases with the extent of the lung involved. Very rapid breathing, cyanosis, liquid dark sputa and low muttering delirium are very bad signs. The absence of leucocytosis is also an unfavourable sign, except in mild cases, though the reverse, viz., its presence, is not necessarily a good sign. Certain complications, *e.g.*, pericarditis, endocarditis, and meningitis, greatly increase the danger.

Treatment.—Many physicians do not recommend any special treatment in the way of drugs or local applications. The patient should be kept in bed, should wear a flannel nightdress slit up the front, so that it can be easily removed for local applications or other purposes, and, where the patient does not object, it is well to place him between blankets. Wrap up the chest in cotton wool, feed him carefully with small quantities of milk and sometimes beef-tea, and watch carefully for any evidence of cardiac failure, or of thrombosis in the pulmonary arteries and right side of the heart. Tincture of strophanthus, in 5 to 10 minim doses every three, four or six hours, is probably one of

the best cardiac tonics to administer, and ammonium carbonate in 5 grain doses is certainly of some use in preventing thrombosis. An indication of the commencement of thrombosis may be obtained by watching for distension of the jugular veins in the neck and by listening at frequent intervals to the second sound in the pulmonary area. Any enfeeblement of this sound is of serious import. Alcoholic and other stimulants should, if possible, be reserved for a later period, when the patient's strength is suffering from the strain of the continuous fever. In alcoholic subjects the use of alcohol at any time is open to question. In non-alcoholic cases, alcohol forms a food as well as a febrifuge remedy. Do not give any of the ordinary antipyretic agents, because the temperature is thereby masked, and it is much safer to keep the fever in check by sponging the skin, by the wet pack or other hydropathic means of treatment. Oxygen is, in our belief, of very great value, provided it is given at an early period. If the funnel by which the gas is discharged be kept at a little distance from the patient's face there is no fear of his inhaling the 80 per cent. of oxygen which Lorraine Smith has found to have an irritant effect in experimentation on animals. The failure of oxygen is, we believe, generally due to its application being delayed till a period when no remedy is likely to be of avail. For delirium, an ice-bag should be applied to the head, and, when necessary, potassium or ammonium bromide should be administered to the patient. Probably saline intra-venous injections for the toxæmia will help in soothing the delirium, and they are certainly worth trying. Leucocyte counts help considerably in discovering how it fares with the patient in his battle with the organism, but, unfortunately, no serum treatment has yet been found to be really beneficial, and upon whose aid we can confidently rely. Once the stage of convalescence is reached, pulmonary gymnastics will be found useful in promoting recovery in the damaged area of lung, and

sometimes painting on iodine is helpful in aiding absorption. There is no question but that Fränkel's pneumococcus is infectious and that certain patients are specially susceptible to an attack, and, therefore, care should be taken not to expose patients with heart disease, influenza, and those who are debilitated, from whatever cause, to the risk of infection.

LOBULAR PNEUMONIA.

SYNONYMS.—The diplococcus lanceolatus may produce an inflammation restricted to one or more small areas corresponding pretty nearly to individual lobules, hence the name *lobular pneumonia*. In most of these cases the inflammation involves also the branches of the bronchi supplying the pneumonic patches, hence the name *broncho-pneumonia* is in frequent use. The inflammation may spread from the lung backwards to the bronchioles, or downwards or outwards from the bronchioles to the lung. In any case it is difficult for the lung tissue to escape when the minute terminal bronchi become inflamed, hence the use of another synonym, viz., *capillary bronchitis*. These three are topographical terms signifying much the same lesions. The brunt of the pneumococcal infection falls upon the cells lining the bronchioles and the lung alveoli respectively. These cells proliferate and desquamate, filling the cavities of the bronchi and of the alveoli, hence there has arisen yet another synonym, viz., that of *catarrhal pneumonia*, a useful term, inasmuch as it focusses attention upon the difference between the character and source of the exudate in the two chief pneumonic conditions caused by the diplococcus lanceolatus. In the graver and more acute form, viz., lobar pneumonia, the exudate is fibrinous and comes from the blood-vessels: in lobular pneumonia, on the other

hand, it is catarrhal and comes from the lining cells of the alveoli and bronchioles, though some albuminous fluid comes from the blood-vessels. This difference in result from the same infection is, like the difference in topographical distribution, an expression of the graver character of the disease in the one affection as compared with the other, but we do not know the reason. It may be due to variations in the germ or in the patient, or in both.

Morbid Anatomy.—The lung shows, both on surface and on section, a number of areas of altered colour and consistence. In size they vary greatly, that of a pea or bean being most typical, but they may be much bigger and may exceptionally run together to form large areas, even to a whole lobe or large part thereof (the pseudo-lobar pneumonia). In position they are seen mostly near the pleural surface, especially towards the root and posterior basal parts of the lung. In colour they vary with age, being red to begin with, and then growing paler until they become of a greyish or greyish-yellow colour. The depth of red varies, the darkest being almost of a purple colour. These are generally smaller than the lighter red areas, and they differ from them also in other respects. Their pleural surface is slightly depressed below the normal level of the pleura, whereas that of the paler red areas is slightly raised. These changes are due to a difference in their method of production. The paler red areas are truly pneumonic from the beginning, and show microscopically an exudate of catarrhal cells with some red and white blood-cells within the lung alveoli; but the darker red areas are not pneumonic to begin with, they are merely collapsed from their supplying bronchi having become blocked by exudate: hence their alveolar cavities, though small and shrunken, are empty, their lining cells swollen but not yet proliferated, and their alveolar capillaries

intensely congested and surrounded by a leucocyte and even red blood-celled exudate. Later, the pneumococcus may reach these areas, when proliferation and desquamation of the epithelium and exudation of fluid follow and they become also pneumonic. The paler shades of colour which are subsequently seen in all pneumonic areas are due to the fatty and other degenerative changes which gradually come on. The lung tissue immediately surrounding the pneumonic or collapsed areas is emphysematous, and this, combined with the generally small size of the patches, makes difficult their detection by percussion and other physical signs.

Symptoms. — The disease may appear in a person apparently healthy (primary form) or in one the subject of pre-existing disease, such as measles, whooping-cough, influenza, and many other diseases, particularly any of the acute fevers. The primary form is almost always seen in children, though a few cases occur in adults. In recent years, owing to the prevalence of influenza, a primary influenzal lobular pneumonia has become comparatively common in adults. The secondary variety is also most frequent in children, though its occurrence in adults is by no means rare. The symptoms, which closely resemble those of lobar pneumonia, are much the same in both varieties. The onset is more sudden in the primary than in the secondary form, and the fever often terminates by crisis in the former, particularly in children, while it is almost, if not always, by lysis in the latter. The chief differences from lobar pneumonia are in the sputum and the physical signs. In children there is rarely any sputum except that brought up by vomiting, and while it is usually present in adults it is not rust-coloured. The physical signs are those of bronchitis, not of consolidation, though patches of this may be detected. Their smaller size, wider distribution, and usually more evanes-

cent character distinguish them from the consolidation of lobar pneumonia.

Diagnosis.—It is distinguished from lobar pneumonia and bronchitis, both of which it may closely resemble, by the character of the fever and by the amount of consolidation. It may be very difficult to differentiate it from early acute pulmonary tuberculosis, and here again the signs of consolidation may help—they appear earlier and clear up faster than in tubercle.

Prognosis.—Lobular pneumonia, whether primary or secondary, whether caused by the pneumococcus or some other organism, is a grave disease, particularly in infants. The feeble and debilitated generally die.

VARIETIES.—This description of lobar and lobular pneumonias is applicable to similar pneumonias caused by other organisms than the diplococcus lanceolatus, for while this germ is the cause of the vast majority (probably over 95 per cent.) of all lobar pneumonias and about 50 per cent. of all lobular pneumonias, other germs may cause similar topographical lesions. A few lobar pneumonias may be caused by the pneumo-bacillus of Friedländer, though this is very doubtful, or by the bacillus coli, or by the streptococcus pyogenes, or even by other germs. A great many lobular pneumonias may be caused by the streptococcus or staphylococcus or bacillus coli among the non-specific germs, or by the influenza, the typhoid, the tubercle or other specific germ. Further, it is common for a combination of germs to be at work, and here again the diplococcus lanceolatus (Fränkel) is the most common coadjutor; it may be combined with any other of the non-specific germs, such as the colon bacillus or the pyogenic cocci, or with any of the specific group, such as the influenza bacillus, the tubercle bacillus, and so on. In these mixed cases the pneumococcus, if present,

is secondary, and the topographical changes in the lung may be caused by it alone, or in combination with the other germ or germs; but however caused, the morbid lesions produced in the lungs, and the symptoms and physical signs to which they give rise, are in most cases similar to those described above under primary pneumococcal infection, though they may be exaggerated or otherwise modified by the general results of the primary infection. The same thing is true of most, if not all, of the acute specific pneumonias, *e.g.*, influenza; but some of the acute non-specific pneumonias present certain differences, both topographically and generally: thus the group of *aspiration pneumonias* (which arise in various conditions, such as necrotic affections of the pharynx, larynx, trachea or bronchi, paralysis of the larynx followed by aspiration of particles of food or irritating secretions), while it causes a lobular pneumonia, is characterised by a more intense local inflammation and greater general disturbance. The exudate is generally more hæmorrhagic or purulent, and distributed over larger areas. The process is apt to terminate in abscess formation or necrosis. *Hypostatic pneumonias* are also examples of intense broncho-pneumonias. They occur in the dependent parts of the lung in persons weakened by antecedent disease. The cardiac weakness gives rise to a more or less intense congestion and œdema of the lung, most marked in the posterior basis, and when one or more of the bacterial irritants reach this congested lung through the bronchi the resulting foci of inflammation are more intense and larger than those occurring in a previously unaltered lung. Histologically they are primarily catarrhal, but owing to the preceding congestion and œdema there may be also much fibrinous and hæmorrhagic exudate.

Treatment.—In cases where the pneumococcus of Frænkel is responsible for the disease, the treatment should

be on the same lines as those already described for lobar pneumonia, but here the patient's distress is often greater, there is more urgent dyspnoea, and therefore steam inhalations and the jacket poultice are of special value. Children have peculiar difficulty with the expectoration, and if the cough is severe and sputum viscid, administer an alkaline cough mixture, such as bicarbonate of soda with squills and senega. Where the bronchial tubes are becoming blocked with sputum and the patient, generally a child, is too apathetic to cough, an emetic is the best method of clearing the stomach and bronchi at one and the same time. The strength must be maintained by the administration of milk, beef-tea, &c., and at an early period there are often indications for the use of alcohol and other diffusible stimulants. Remember the risk of œdema of the lungs, and in very young children it is desirable to change the child's posture in bed, and even occasionally to have the child taken up and held in its nurse's arms before the fire. Tincture of strophanthus or other cardiac tonic is usually necessary, and there is no disease in which we believe oxygen inhalations will be found more beneficial if administered sufficiently early and with due care. The jacket poultice should not be kept on too long or used too often for fear of depression. The temperature must be kept in check, and this should be accomplished by cold sponging, cold baths or similar means; but the antipyrin group of remedies should not be used. Once the stage of convalescence is reached, tonics, such as the syrup of the iodide of iron, cod-liver oil and malt should be ordered, and change of air is important. This description of the treatment suitable for lobular pneumonia is specially applicable to children, but very similar methods may be followed in the case of adults.

Two types of pneumonia included in this group require separate reference. In *aspiration pneumonia* the physician has too often a hopeless task, and generally a fatal result

rapidly ensues. Try to prevent the lung from becoming gangrenous, and possibly antiseptic inhalations may be helpful, but the chief point is to maintain the patient's strength. When treating a *hypostatic pneumonia*, stimulate with cardiac tonics and such remedies as spiritus etheris, spiritus chloroformi, and spiritus ammonii aromatici; 20 minims of each of these forms an excellent mixture. The head should be kept as high as possible, and the patient's position in bed altered from time to time.

PURULENT PNEUMONIA AND ABSCESS OF THE LUNG.

Causes.—Purulent pneumonia signifies a pneumonia with purulent lymph or pus diffusely spread through the inflamed areas: abscess of the lung signifies liquid pus localised to a definite cavity or cavities. Either may arise as a terminal process in an intense lobar or lobular pneumonia, particularly the latter, or as a secondary infection from an injury or some necrotic focus elsewhere in the body. In that case they are mostly caused by a streptococcus or staphylococcus pyogenes, but may be produced by the pneumococcus acting alone. Aspiration pneumonias often result in a purulent pneumonia or abscess formation, and among the pyæmic processes causing the same result are malignant endocarditis, puerperal infections, and osteomyelitis. When the emboli are minute and widely spread, purulent pneumonia is more likely to follow: when they are large, suppurative infarcts or abscesses result. Since the pleura is almost certain to be implicated in these cases, a resulting pleurisy, often purulent, will follow. Such an empyema may be encysted. While this is the commoner method

of evolution of these lesions, an opposite course may be seen, viz., a purulent pleurisy to begin with and a resultant purulent pneumonia or abscess to follow. In this case the lung is infected by direct perforation or through the pleural lymphatics and only its outer parts affected, less commonly its whole thickness. Similarly, a pulmonary abscess may arise from a subphrenic abscess, a liver abscess, a mediastinal abscess or other suppurative process external to the lung. In all cases of either purulent pneumonia or abscess of the lung the probable occurrence of associated purulent inflammations elsewhere, e.g., in the endocardium, pericardium, brain and its membranes, must be remembered.

Morbid Anatomy.—There is usually more than one abscess. The common size is about that of a hazel nut, but it may be as small as a pea or as large as an orange. The walls are hæmorrhagic, œdematous, shreddy or gangrenous, or composed of a smooth firm wall.

Symptoms.—They resemble those of simple pneumonias, but those due to the general septic infection may be very intense and overshadow all others. Purulent pneumonias often run a rapid course, but abscess may be considerably prolonged. The pus is almost certain to make its way into a bronchus and to be expectorated in large amount. The physical signs may be very unsatisfactory and indefinite, but the hectic fever, the troublesome (often paroxysmal) cough, and the excessive purulent expectoration at intervals are very suggestive. The signs of a cavity may be got after such an evacuation, *vide* page 580. The breath or the sputa may be foetid or suggest the smell of new mown hay, or the sputa may be hæmorrhagic or mixed with brownish shreds of lung tissue. These cases of dark brown offensive sputa are the most serious, as they indicate a gangrenous process.

Diagnosis.—Bronchiectasis is the most difficult disease to separate from abscess of the lung. Sometimes it cannot be done. In both cases the sputum may be foetid and abundant, but it is usually more abundant in bronchiectasis than in abscess; moreover, the former disease usually runs a much longer course than does abscess, and usually gives more distinctive physical signs. A loculated empyema, especially if deeply seated between the lobes of the lung, is most difficult to distinguish from abscess, but, when it most resembles it, viz., when it ruptures into a bronchus, it becomes unnecessary to do so, because it becomes virtually an abscess of the lung.

Prognosis.—It is grave in all cases. The most favourable cases are those in which there is a single abscess situated near the surface, which can be thoroughly emptied by operation. Spontaneous evacuation into a bronchus may also lead to contraction and cure, but the evacuation is apt to be very imperfect and the suppurative process to continue until the patient is worn out or carried off by some associated lesion elsewhere.

Treatment.—Keep up the patient's strength by feeding with milk, beef-tea, &c., so as to assist Nature in the struggle with the organisms and their poisons. Cardiac tonics, alcohol, and other diffusible stimulants are generally required at an early stage in the disease. Later, try the treatment for Bronchiectasis (see page 513), and the question of operative interference must be carefully considered where there is a localised abscess. In many of these cases it offers the only hope for the patient. There is one remedy to which special reference should be made. Quinine may be freely administered. It will much more probably counteract the toxins than will any other drug; in some cases antistreptococcic serum might be tried. It is hardly necessary to state that where an extensive

area of lobar pneumonia is undergoing suppurative change no treatment is likely to prove of any avail.

GANGRENE OF THE LUNG.

Causes.—Gangrene of the lung is always a secondary condition. It results when organisms capable of causing putrefaction reach necrotic pulmonary tissue. Its evolution thus closely resembles that of abscess. It may follow upon intense lobar or lobular inflammation, brought about in the usual way, or upon embolism, or upon direct extension from outside, *e.g.*, the ribs, the subdiaphragmatic structures, the œsophagus, &c. Aspiration and bronchial compression pneumonias must be specially mentioned. In all gangrenous processes of the mouth or respiratory passages, necrotic material may reach the lungs and set up an intense inflammation, going on to gangrene. Mediastinal tumors, aneurisms, &c., may press upon a bronchus, or a bronchus may become plugged by necrosed tissue or by a foreign body, such as a false tooth or a coin. Collapse and intense congestion follow in the affected part of the lung, which, if the germs of inflammation and putrefaction reach it, may undergo intense inflammation, necrosis and gangrene. In the same way, all devitalising states, such as the acute fevers, starvation, and wasting diseases, may allow of similar congestions, inflammations and gangrenes. The precise condition will depend upon the double factors, *viz.*, the resisting power of the patient and the nature and virulence of the attacking germs. In one case it may be a simple pneumonia, in another a purulent one, and in another a gangrene. Diabetes must be specially mentioned as a devitalising disease which is very liable to be followed by pulmonary inflammations and gangrene. Embolic and direct extension gangrenes need no explanation.

Morbid Anatomy.—There are two varieties—a circumscribed and a diffuse. The former occurs in well-defined areas of a small or large size. They present marked changes in colour and consistence, which vary somewhat according to their duration. In colour they are intensely red at first, generally becoming darker later, reaching a dark brown or even black or greenish-black colour: occasionally they get lighter and even purulent looking. In consistence the gangrenous part may at first be firmer than normal, but later it becomes softer and breaks down into a pulpy mass or fluid material with a putrid odour. The blood-vessels and bronchi may remain and pass through the cavity or they may break down. The walls are usually shreddy and surrounded by an intensely congested and œdematous lung tissue, often in a state of consolidation. The diffuse form is not so sharply defined. It is more widespread, involving, it may be, a large part of one or more lobes. The colour is similar but the softening is rarely so great, except at times, in small foci throughout the gangrenous area. The relations to the pleura and other organs are similar to those of purulent pneumonia and abscess.

Symptoms.—In addition to the symptoms and signs of pneumonia, special attention has to be paid to the sputa and to the occurrence of hæmoptysis. The latter may be very severe and frequent. It would occur even oftener if rupture of the vessels were not preceded by thrombosis. The sputa are always diffluent, mucopurulent, purulent, greyish, brownish, or of a prune-juice or chocolate colour, and intensely foetid. There is generally intolerable foetor of the breath. It may be a very early symptom and may be so great, especially after a fit of coughing, as to be noticed the moment the patient's room is entered. It is sometimes intermittent, but is rarely absent, except occasionally in the insane, in diabetic and in embolic cases.

Prognosis.—Recovery is rare. Death occurs most frequently before the end of the second week and is rarely delayed as long as the second month.

Treatment.—Support the patient's strength in every possible way and thus strive to put him in a position to resist the toxins to which he is exposed. Vaporise such antiseptics as carbolic acid, creosote, or guaiacol, or spray them (suitably diluted) in such a way that the patient may inhale the antiseptic into the lungs. This treatment is necessary so as to render the duties of the attendants possible in cases where, as too often occurs, the odour of the breath is very putrescent. The direct injection of antiseptics into the lung has been recommended, and iodoform has been so used, but there is always the risk that when the needle is withdrawn it may leave a septic track which may give trouble. Operative interference has sometimes proved satisfactory.

CHRONIC NON-SPECIFIC PNEUMONIAS.

Causes and Morbid Anatomy.—These result in fibrous tissue formation within the lung, constituting fibrous or interstitial pneumonias. Several varieties have been described:—(1) A lobar form, where a whole lobe or its greater part has its alveolar and peribronchial tissue generally thickened. It may arise independently of acute lobar pneumonia or follow after it, when the name "sub-acute indurative pneumonia" has been applied to it by some. (2) A broncho-pneumonic form, which is much more frequent, being particularly common in children; in it the fibrous tissue is not diffuse but restricted to patches scattered throughout the lung. It may be primary, but in many cases it follows upon acute lobular pneumonia,

or upon bronchiectasis. (3) A pleurogenic form, in which dense trabeculæ of fibrous tissue pass inwards into the lung, even to its root, from the densely thickened pleura. (4) An inhalation form, the *pneumonoconiosis*, i.e., the form of chronic patchy pneumonia set up by the constant inhalation of dust. Inhaled dust particles are usually caught by the mucus of the respiratory canals and expectorated. It is only when they are very numerous that some escape and reach the pulmonary alveoli. There they set up a certain amount of catarrh and may still be expectorated. Failing this, they may reach the alveolar lymphatics by either directly penetrating the alveolar walls or by being carried by cells through the osteoles. They are carried by the lymphatic stream to the peribronchial and perivascular lymphatics, and thence to the bronchial glands at the root of the lung, or along the interlobular septa to the visceral pleura (its deeper layers). They may collect in little clusters anywhere along these paths. When they do so, they become surrounded by fibrous tissue, which will be greater in amount the greater the number of the particles and the more mechanically irritating their character. These fibrous foci vary in size from minute specks to quite large areas. The largest are generally due to the confluence of small ones. The inhaled dust particles also impart a characteristic pigmentation to the areas. Individuals who have dwelt for a considerable period in cities inhale a large quantity of dust, chiefly soot, and their lungs consequently show a certain amount of black pigmentation. It is not considered to be abnormal unless it reaches a considerable degree, not likely to be acquired except by workers in certain trades. Thus coal miners inhale much unburnt carbon dust and their lungs show numerous small shot-like areas scattered throughout the lung substance, particularly in the upper lobe and in the deeper layers of the pleura. They may be so close together as to convert a

large area of the lung into a seemingly uniform solid black mass. The bronchial glands are enlarged and pigmented, the bronchi congested, and the pleura chronically thickened and adherent. This condition of lung is spoken of as *anthracosis*. In the same way, the lungs of workers in stone or metals or clay become similarly affected, giving a condition known as *silicosis* or *chalicosis*, *siderosis*, or *aluminosis* respectively. The stone and metal particles are more irritating than the others, and the lungs are correspondingly more markedly affected. The stone particles impart a grey pigmentation to the patches, the iron oxide particles a red or black pigmentation, but in all cases the patches tend to become more or less pronouncedly black, because of an admixture with carbon. What is the explanation of the formation of the fibrous tissue in these fibrous pneumonias? In the lobar, lobular, and bronchiectatic varieties it is the result of the action of some one or other of the germs of inflammation, but in the inhalation forms how do the inhaled solid particles produce it? If they are soluble, as they sometimes are—*e.g.*, mercury, arsenic—it is easy to understand how they may be dissolved, producing solutions which act upon the tissues, much as bacterial toxins do; but in the case of the many—*e.g.*, carbon, quartz, iron, brass, clay, &c.—which are insoluble, the same explanation cannot be given. The most probable explanation is that the inhaled particles of dust, while mechanically capable of a certain amount of tissue destruction, have germs adherent to them, to which, and not to the dust particles themselves, the subsequent irritation and resulting fibrous tissue formation is due. Tubercle may be carried in in this way, and the formation of cavities, which is sometimes met with in inhalation pneumonias, is probably due to a coincident or subsequent infection with the tubercle bacillus.

Symptoms.—When cough, pain in the chest, shortness of breath, dulness or diminished chest movement persist

for a long time after the acute pneumonia disappears, this chronic pneumonia may be suspected. The primary and inhalation forms closely resemble bronchitis, but the character of the sputum, diminished movements or contraction of the chest on one side, and the severe dyspnoea, may enable a diagnosis to be made. Bacteriological examination of the sputum is necessary, as many cases simulate chronic phthisis.

Treatment.—Few interstitial pneumonias admit of much special treatment because the lung is damaged to a greater or less extent, and too often that damage cannot be repaired. In all cases the patient's strength should be maintained, and in most diseases belonging to this group cod-liver oil in some form is one of the most important medicines, while the question of climate is frequently a matter calling for careful consideration. Many subjects of interstitial pneumonia winter in Egypt or in some other dry and warm climate without suffering from any attacks of bronchitis which is such a common sequel to the disease. Counter-irritation with iodine is frequently of value in the pleurogenous type of interstitial pneumonia.

In the fourth type mentioned above, commonly called pneumoconiosis, and which includes dust diseases of all kinds affecting the lungs, the treatment depends much on the nature of the dust, whether irritating, and therefore likely to produce destruction of lung, or not. The treatment for bronchitis and, where there is cavity formation, the treatment for bronchiectasis are suitable, while if tubercle is superadded, the treatment for chronic pulmonary tuberculosis is appropriate. The prophylaxis of dust diseases is of great importance, and much more care is now taken to obviate the amount of dust amongst which the workers of bygone days had to spend their working hours. This has been accomplished by ventilation and other means, and where dust is inevitable, and is

peculiarly irritating, as in the case of steel dust, masks are worn by the workers. It is impossible to assume that even coal dust is not objectionable, because, whatever antiseptic properties carbon may possess, it certainly interferes seriously with the pulmonary lymphatics, and therefore prepares the way for the advent of tubercle.

ACUTE SPECIFIC PNEUMONIAS.

It has already been mentioned that certain specific germs—*e.g.*, diphtheria, typhoid, influenza, tubercle, glanders—may, acting along with the *diplococcus lanceolatus* of Fränkel, or with any of the inflammatory cocci, cause a pneumonia either of the lobar or lobular form, but certain of these germs, notably the typhoid, influenza and tubercle, may by themselves produce a pneumonia. The two first require only a short notice.

TYPHOID PNEUMONIA.

The respiratory troubles in typhoid fever have already been referred to. Inflammation of any part of the respiratory tubes—*e.g.*, larynx, bronchi (bronchitis is a common early symptom of typhoid)—or of the lungs is of frequent occurrence. Both lobar and lobular pneumonias occur as initial manifestations. The former is particularly important, as the case has at first all the appearances of an ordinary lobar pneumonia and the typhoid fever may remain unsuspected till the second week, when, instead of the usual pneumonic crisis, the true symptoms of typhoid gradually develop. It is not yet known from actual bacteriological observations whether the pneumonia is due to the pneumococcus or to the typhoid bacillus, but general considerations lead us to suspect the

former rather than the latter. The same thing is probably true also of the initial bronchitis and lobular pneumonia of typhoid. Both forms of pneumonia are met with as complications occurring later in the disease, about the third week. The lobular and pseudo-lobar forms are more frequent than the true lobar. Many bacteriological observations have been made upon them, and it is said that the typhoid bacillus can be obtained from the lung with ease in pure culture by inserting a hypodermic needle into the dull area. This can be done without risk, and has been suggested as an additional method of diagnosis in doubtful cases. It would thus appear that some at any rate of the later typhoid pneumonias are due to the local action of the typhoid bacilli in the lung.

INFLUENZAL PNEUMONIA.

A pneumonia occurring in the course of influenza is of great frequency. Prior to the appearance of influenza in this country (1889) it was generally held that lobular pneumonia was almost confined to children. It was rarely met with in adults. This is no longer the case. Influenza is responsible for the change. It is not the influenza bacillus, however, to which the pneumonia is due, at least not directly, for the majority even of influenzal pneumonias are actually caused by the pneumococcus. The toxin of the influenza bacillus weakens the resisting powers of the body and so renders the lung tissues susceptible to the pneumococcal attack. Both lobar and lobular pneumonias, particularly the latter, are met with in influenza, and the former are probably always caused by the pneumococcus. Many of the latter are also caused by it, either in combination with the influenza bacillus or alone, but a good many are due to the direct local action of the influenza bacillus itself. It is found in great numbers in the mucus and in the leucocytes in the lumen of the

bronchi. It penetrates the epithelium of the bronchi and sets up inflammation of the submucous coat, showing itself by a marked emigration of leucocytes and by a serous exudate into the bronchial walls and their lumen. The inflammation spreads outwards, and in the case of the finer bronchi, whose walls are thin, it soon reaches the surrounding lung parenchyma, causing pneumonia. This pneumonia is thus patchy or lobular, and it is catarrhal in so far as it causes desquamation of the epithelial cells lining the alveoli; but there is also a marked exudate from the blood-vessels, which may be entirely serous or partly fibrinous, and which usually contains emigrated leucocytes in large numbers. This leucocyte emigration is sometimes a very marked feature, and foci of purulent softening may be formed, in which the influenza bacillus has been found in pure culture. Pulmonary gangrene may occasionally supervene. It has sometimes been said that there is something special about influenzal pneumonias, but this is not the case. They owe their gravity not to the local lung lesions but to the toxins which are manufactured in these lesions. These may be influenzal or pneumococcal toxins alone or combined, in all cases powerful cardiac depressants, and it is probably this accumulation of the cardiac poison, rather than the embarrassment caused by the pulmonary lesions, which determines the cardiac failure which is of such frequent and often fatal occurrence in influenzal cases. The pulmonary lesions are important for another and altogether different reason, viz., the increased susceptibility to tubercular infection. The patient must be carefully protected against this, particularly in cases where the pulmonary disease does not entirely clear up but leaves some chronic lesion. The signs of this may be very indefinite, but it should be suspected where recovery is not complete. There is some cough, and influenza bacilli may be detected in the sputum for many weeks after the acute disease has disappeared. If

tubercular infection supervene in such cases, the downward course is generally rapid.

PULMONARY TUBERCULOSIS.

The tubercle bacillus may enter the lung by—(1) The inspired air (aerogenic tubercle); (2) the blood-stream (hæmogenic tubercle); or (3) the lymph-stream (lymphogenic tubercle).

ACUTE MILIARY TUBERCULOSIS.

Morbid Anatomy.—In the last two the form of tuberculosis produced is generally the acute miliary, the minute grey tubercles in the hæmogenic form being scattered everywhere through the lung substance, looking as if it had been indiscriminately peppered with them; while in the lymphogenic form they are more or less restricted to certain parts of the lymph channels, being seen particularly in the deeper layers of the pleura, the interlobular septa, around the bronchi and larger blood-vessels, and in the bronchial glands. The rest of the lung substance is congested, sometimes deeply, but is not consolidated. There may be a certain amount of vesicular emphysema around the tubercles, and this is often very marked and widely distributed in children. There is generally also marked bronchitis, and even small ulcers in the finer bronchi, formed by the miliary tubercles in their walls becoming caseous and discharging their contents into the lumen. The tubercles in the lung substance are generally more minute, as well as more numerous and more diffusely distributed, in the hæmogenic than in the lymphogenic forms. They vary in size from minute translucent specks when youngest, hardly visible to the unaided eye, to large pin-head or nearly pea-sized opaque greyish nodules when they

are older and of slower formation. They are very irregular in shape, and are formed in the interalveolar, interlobular and other septa, and the older ones usually show caseous centres with giant, endothelial and round cells at their peripheries. Caseation and giant cells are usually both absent in the tubercles of most acute formation. The neighbouring alveoli are compressed, and usually show a varying amount of mixed catarrhal, fibrinous, round-celled and hæmorrhagic exudate, so that the lesions constitute a mixture of *interstitial and catarrhal or lobular pneumonia*. There is never any reactive fibrous tissue formation at the periphery of these acute miliary tubercles; but in the chronic miliary tubercles, which are of much more rare occurrence, it may be considerable. The tubercle bacilli enter the blood-stream either from a pre-existing tubercular lesion, such as an old caseous focus in the lung (generally somewhere in the apex) or in a cervical, bronchial or other lymph gland or in a bone, or they may enter it through a mucous or endothelial membrane, such as the bronchus or intestine or pulmonary alveolar wall, without creating any visible lesion at the point of entrance. The entrance into the blood-stream may be direct, as in ulceration into a vein, or indirect, as through the lymph-stream reaching the ~~vein~~ by the thoracic duct, as in tuberculosis of the ~~lymph~~ glands. Many believe that the numbers of the failure reaching the venous blood at a time in this ~~latence~~ are small and so are able to cause only the chronic ~~portant~~ of miliary tuberculosis of the lung. The tubercle ~~by~~ the may enter the lymph-stream either from the pleura or ~~the~~ bronchial and other lymph glands, but fail to enter ~~the~~ blood-stream.

Associated Lesions in other parts of the Body.—It is rare that the miliary tubercles are present in the lungs alone, though they are always more numerous and generally larger there than elsewhere. They are mostly widely distributed throughout the body, particularly in the spleen, liver, kidney,

bone-marrow, and serous membranes, notably the meninges of the brain. But whether they are visible in these organs or not there are always signs of a general toxæmia, viz., cloudy swelling or even fatty degeneration of the heart, liver and kidneys.

Symptoms.—The onset may be sudden or insidious. The latter is often seen in children, where, also, there may be a preceding attack of measles or whooping-cough. The symptoms upon which most reliance is to be placed are the temperature, the cough, dyspnœa and cyanosis. The temperature rises to about 102°F. and keeps up, the evening being slightly higher than the morning temperature. Occasionally an inverse type is seen, in which the morning temperature is considerably the higher. The pulse is generally rapid. There is almost always cough, though in exceptional cases it is slight or absent. Expectoration is often absent, and when present is slight, the sputa rarely showing tubercle bacilli. Dyspnœa is one of the earliest and most constant symptoms. It is often greater than the physical signs seem to warrant. Cyanosis of the lips and nails is frequently present, and is sometimes very marked. Prostration appears sooner or later and becomes marked towards the end, when it passes into the “typhoid” state or cerebral symptoms supervene.

PHYSICAL SIGNS.—Sibilant and sonorous ronchi are generally heard all over the chest, and so loudly as to obscure other signs. Fine crepitations may be present, and larger rales usually appear late in the disease.

Diagnosis.—In the early stages it is particularly difficult, as the symptoms and signs are so like those of early typhoid fever or bronchitis. Ophthalmoscopic examination may reveal the presence of miliary tubercles of the choroid. The Widal test may help to distinguish it from typhoid, as does also the size of the spleen, for though this organ is

enlarged in both diseases, the enlargement is greater in typhoid. There may also be a history of pre-existing tubercle in the bones or elsewhere.

Prognosis.—It is always grave. Death may follow in a week, or not for about three months. The average duration is said to be about five weeks. The symptoms may almost completely subside for a time, but generally break out again, though some few cases recover altogether.

Treatment.—Treat the symptoms as they occur and support the patient's strength. Keep down the temperature by cold sponging, sometimes by the use of ice, or by other hydropathic methods. Quinine is a useful remedy with which to check the excessive sweating. The constipation, cough, diarrhoea, and other symptoms should be treated as they occur.

ACUTE AEROGENIC OR BRONCHIOGENIC TUBERCLE.

This form of infection is much the commonest and results in more marked pneumonia, mainly of the broncho-pneumonic type, and owing to the extensive caseation which occurs it is often called *acute caseous tuberculosis* or "*acute caseous broncho-pneumonic tuberculosis*," or, owing to the great destruction of lung tissue which it produces, the names *acute phthisis*, or *acute pneumonic phthisis*, or *phthisis florida*, or *galloping consumption* are often used. It is commoner in children, in adolescents, and in young than in other adults.

Morbid Anatomy.—In a typical case the lung shows everywhere a number (generally a large number) of white, greyish or yellowish-white caseous-looking masses or tubercles of irregular size and varying shape scattered through its substance, but, while the distribution of the masses may seem at first to be indefinite, closer examination shows that they are arranged around the bronchi,

particularly the smaller bronchi, and some trace of the lumen can often be found in their centre. Their bronchial distribution accounts for their more frequent occurrence towards the pleural surface, and for their irregular size and shape. The size may be quite small, almost miliary, when the growth has not passed much beyond the walls of a bronchus or a few lung alveoli; even then it is caseous, and it is uncommon for typical translucent miliary tubercles to be seen. On the other hand, it may be of considerable size when it involves many alveoli around the bronchus, and particularly when several such areas coalesce together. A large part of a lobe may thus be rendered fairly solid. The shape is characteristically irregular, the periphery often showing the appearances presented by the contour of a bunch of grapes—much like what one would expect if a semi-solid substance injected into a small bronchus were to fill the lung lobule in whole or part and pass beyond its periphery at places into contiguous alveoli; for though an artificial injection of the bronchus would not behave in this way, it is pretty closely what happens in this disease, since it destroys the alveolar wall in many places. The rest of the lung substance is deeply congested, as are also the bronchi (the general bronchitis being often intense). The pleura is almost always involved. The caseation which is so characteristic a feature of all the masses spreads into the pleural tissues, which become of a greyish or yellowish-white colour in the implicated area and usually give way, allowing of some of the contents of the caseous mass escaping into the pleural cavity. The pleura around the necrotic part becomes deeply congested and covered with a fibrinous exudate, which in some cases may spread over the necrosed part and protect the pleural cavity for a time, but not for long, except in the rarely occurring slow cases, where the reactive inflammation in the pleura may have time to result in fibrous tissue formation, which may

successfully seal up the weak spot. Usually the contents escape into the pleura and set up an acute tubercular pleurisy, though in some cases—*e.g.*, where the caseous foci have had time to become contaminated with other germs—the resulting pleurisy may be simple or suppurative. There is one other lesion in the lung itself which is nearly always present, and which must be carefully looked for, and that is a chronic tubercular one. It is mostly situated in the apex, and consists of a caseous mass or cavity or other chronic tubercular lesion more or less walled in. It is the result of a tubercular infection occurring long prior to the present disease and remaining more or less quiescent until now, when lessened resistance on the part of the patient or some other cause has enabled the tubercle bacilli resident therein to break beyond their barriers and spread to form the acute broncho-pneumonic lesions above described as characteristic of the disease. This completes the macroscopic picture presented by the morbid anatomy of the disease except for two variations which are sometimes seen, *viz.*, extensive consolidation and cavity formation. The former may arise from the rapid spread and coalescence of separate lobular areas or from a rapid and extensive formation of the caseous pneumonia, which occasionally assumes a massive or lobar form, when it presents appearances very like those of grey hepatisation. The latter (cavity formation) is usually seen in the form of several small cavities (rarely larger than a hazel nut) replacing some of the caseous masses. The caseous masses vary in consistence like soft or hard cheese, and they may, the softer ones particularly, break down and be discharged through the bronchi as sputa. The walls of the cavities are thus soft, crumbly and irregular, being formed by the caseous pulmonary tissue. The walls of the bronchial tubes and the vessels are more resistant than the parenchyma of the lung to the caseous action of the tubercle, and these tubes may be

seen in some instances to pass through such a cavity from wall to wall, though in most cases they break down also. The expectorated caseous material sets up the intense bronchitis already mentioned as frequently present, and, further, other germs, *e.g.*, the pyogenic organisms, may now reach the interior of the cavities by means of the inspired air and thus set up a mixed infection. The microscope shows the caseous lesions to consist of structureless granular material surrounded by a varying amount of round cells mixed with some epithelioid cells. Giant cells are not seen except in the more slowly produced lesions. Tubercle bacilli can be made out in the caseous material, particularly towards its margin, where they are generally present in large numbers. The neighbouring lung alveoli are partly compressed and partly filled with catarrhal, leucocyte or fibrinous exudate. In the early stages the bronchus can be seen towards the centre in a state of acute bronchitis, later, when caseation is sufficiently advanced, it may entirely disappear. The evolution of the lesions follows two main lines. In the one the caseous material from the original chronic lesion may ulcerate into a bronchus, and though part may be expectorated part may be moved to and fro in the bronchi and aspirated into neighbouring and fresh bronchi, and thus reach fresh lung alveoli, when each acting as a new centre gives rise to fresh tubercular growths. The primary or any of the secondary caseous foci may eat into a blood-vessel and cause hæmogenic tubercle, in which case miliary tubercles may be found in the lungs and other organs. This sometimes occurs early, and often occurs late, in the course of the disease. The other main line of evolution is that which occurs when the primary focus itself is formed, or when the disease begins without such a pre-existing primary focus. In it the tubercle bacilli, probably in a high degree of virulence, reach the mucous membrane of the bronchi and epithelium of the

lung in a very susceptible person, lodge therein, multiply, and cause an acute bronchitis, peri-bronchitis, and a mixture of catarrhal and interstitial pneumonia. There is swelling and multiplication of the endothelium of the interalveolar capillaries, round-celled and serous exudation into the interalveolar septa, proliferation and swelling of some of their connective tissue corpuscles, proliferation and desquamation of the alveolar endothelium, leucocyte, serous and fibrinous exudation into the alveoli in varying amount and degree; but the most characteristic change of all has yet to appear. It does so sometimes soon, sometimes late, and consists in *caseation*. It appears first about the centre of each lesion and quickly spreads, and as fast as the interstitial and parenchymatous pneumonic process extends at the periphery, as fast does the process of caseation radiate outwards from the centre. It generally follows upon the cellular and exudative changes too rapidly to allow of the formation of typical giant cells. This method of formation prevails in all the primary lesions, and a practically similar one in the secondary lesions. In the latter there is this difference only, that the tubercle bacilli are concentrated by being held together by the caseous material aspirated at the same time. They thus secure a lodgment more rapidly and exert their action all the more quickly because the caseous material contains pre-formed toxins as well as bacilli. Looked at in this way, there is but one main line of evolution, the one being a further process of the other. The nature of the process may be summed up thus—an acute bronchitis, peri-bronchitis, interstitial and parenchymatous lobular or patchy pneumonia, in which caseation rapidly supervenes.

Symptoms.—The onset may be sudden or gradual (more common). Some of the suddenly beginning cases present the typical symptoms and signs of acute lobar pneumonia, from which it may be impossible to separate

them until after the lapse of a week or two. The presence of the pneumococcus alone in the sputum is not sufficient to differentiate them, for it is probable that these cases illustrate a double pneumococcal and tubercular infection, each organism favouring the action of the other, and it is only when the pneumococcal pneumonia subsides and clears up that the tubercular one can be detected. Other cases begin, like typhoid fever, with prostration, anorexia, fever, &c., and though the course of the fever may differ somewhat from that of typhoid, it does not always do so. But whether the onset is sudden or gradual, careful enquiry may reveal the presence of previous ill health, particularly of cough, as is to be expected from what has been said under morbid anatomy as to the frequent existence of some chronic tubercular focus. The symptoms characteristic of the disease are due partly to the lung lesions and partly to the action of the tubercular toxins upon the body generally. The former cause cough, hæmoptysis, dyspnœa and cyanosis, the latter cause fever, restlessness, weakness, progressive emaciation, and sometimes also enlargement of the spleen and albuminuria. The cough is usually frequent and irritating (often described as hacking). The sputa are mucous at first, but soon become opaque or purulent. They generally show tubercle bacilli in large numbers. The hæmoptysis may be the first symptom. It may be slight, frequent or severe. Both the dyspnœa and the cyanosis are usually moderate. The fever is usually about 2°F. higher in the afternoons or evenings than in the mornings. The restlessness produces sleeplessness and, later, even delirium. The physical signs commence with those of bronchitis, and are followed by those of patchy consolidation and then cavity formation.

Diagnosis.—The separation of this affection from acute lobar pneumonia and from typhoid fever may present

at first the chief difficulty, but the future progress generally puts aside all doubt.

Prognosis.—Most cases progress steadily downwards, the average duration of life, after the case is sufficiently advanced to permit of a definite diagnosis, being about two to three months. Some cases become chronic and some may be arrested, at any rate for a time if not altogether.

Treatment.—The disease is often appalling in the rapidity of its progress, and therefore it affords little chance of successful treatment. Support the patient's strength by milk, beef-tea, egg-flip and other nourishing articles of diet; aid the heart's action by the use of strophanthus or other cardiac tonic; and keep fever in check by sponging and quinine. Belladonna not merely aids in soothing restlessness, but limits the excessive sweating. The cough may require the use of sedatives, and opium may have to be administered. Hæmorrhage may necessitate the treatment already prescribed on page 531, but generally it is not serious and does not call for much interference.

CHRONIC SPECIFIC PNEUMONIAS.

The most important of these is the tubercular. It constitutes the disease known as chronic aerogenic or bronchogenic tubercle, or more commonly as chronic pulmonary tuberculosis or chronic phthisis.

CHRONIC PULMONARY TUBERCULOSIS OR CHRONIC PHTHISIS.

Evolution and Morbid Anatomy.—The tubercle bacilli set up, at the beginning, similar changes to those of the acute form, but the progress is slower, chiefly because the

resisting powers of the patient are greater. The first lesion is either a catarrhal exudate in one or more alveoli or the formation of a minute tubercle in the interalveolar septa, or a combination of both these lesions. It enlarges by a peripheral extension, thus gradually involving a larger patch of lung tissue. The virulence of the infecting bacilli may have something to do with the rate of progress, but it is the suitability of the soil rather than the vitality of the seed which is the chief factor. There is no hard and fast line between acute and chronic forms. They run into one another, but the great majority of all cases of pulmonary tubercle run a prolonged course of many years, and are thus undoubtedly chronic. In them, the bacilli set up a pneumonic focus, or it may be more than one. The apex of one lung is its commonest site, but towards the root is also common, and it may appear elsewhere. Caseation usually follows the pneumonia after a time and a caseous patch of varying size is formed, at the periphery of which giant-celled tubercles are generally clearly seen lying in and surrounded by a considerable amount of fibrous tissue. The neighbouring alveoli are compressed and partly pneumonic, showing sero-fibrinous or catarrhal exudate, while the interalveolar septa are thickened and may show tiny tubercular foci (illustrating local lymphatic extension) at some little distance from the caseous mass. The tubercle bacilli thus cause a giant-celled and caseous pneumonia (partly interstitial and partly parenchymatous), and the connective tissues of the lung produce a reactive fibrous tissue formation which tends to encapsulate the tubercular focus, and thus to retard or prevent its further growth. There is much variety in the proportion in which these two changes of caseation and fibrosis occur, and the rate and character of the progress of the disease depends upon which of them is securing the mastery: when it is the caseation the disease spreads; when it is the fibrosis the disease is retarded. In most cases the primary caseous

focus reaches only a very small size before peripheral fibrosis begins; but the bacilli and their toxins invade this fibrous wall and cause caseation in it in turn. In this way, whether much or little fibrosis occurs, the caseous centre may gradually grow, involving more and more alveoli and their septa until it reaches a considerable size. It rarely spreads equally in every direction, hence it is irregular in shape when it reaches any size. It is incompletely walled in by dense fibrous tissue of unequal thickness, when completely walled in the disease may become quiescent. It may remain quiescent for a long time, and it may even die out, when the contained caseous mass dries up and may have lime salts deposited in it, forming a gritty mass or a calcareous nodule. It is impossible to say how long tubercle bacilli will live imprisoned in this way, certainly for many months, and it may be even for several years. This has to be borne in mind in speaking of recovery in cases in which tubercle bacilli disappear from the sputum and active pulmonary signs cease, which, in short, appear to be completely restored to health. That a certain number of cases completely recover there can be no doubt, from the frequency with which old and dead tubercular foci are found in persons dying from other diseases. Extensive statistics have been published, and the proportion of such cases accidentally discovered post-mortem is put at varying figures from ten to twenty per cent. of all cases of fatal disease. It would not be safe to say that Nature cures this percentage of all cases of chronic pulmonary tuberculosis; but it proves indubitably that a cure is frequent. Moreover, the slow progress of the disease in its earlier stages—the great resistance exerted by the pulmonary tissue against its spread—obviously suggests that if that resistance could be increased, even by ever so little, the same happy result might be secured in many, if not all, cases. Therefore we seek to strengthen and increase the vitality of the patient. This is the basis of the present day

enlightened treatment of phthisis. The disease may break out afresh after a long period of quiescence, but whether this is due to a fresh infection or to the former lesion lighting up it is impossible to say. When it does break out after quiescence, or when it continues to spread without resting, the process is generally the same. The lung tissue surrounding the mass becomes pneumonic and the caseation spreads into it in turn. These new tubercular formations may become merged with the primary mass or remain separate, at least for a time, growing themselves exactly in the same way as the first, and showing the same caseous and fibroid changes. The centre of the caseous mass tends sooner or later to soften and discharge its contents into a neighbouring bronchus and be coughed up. A cavity is thus formed, as in the acute cases, at first small but tending to get gradually larger by the surrounding caseous material breaking down and escaping. Such cavities are of all sizes, from that of a pea or a bean to that of an orange or even bigger, hollowing out the greater part of a lobe. The lining walls are shreddy and necrosed or smooth, according to the age of the cavities (the older they are the smoother they become), and are bounded outside by dense fibrous tissue. The walls of the bronchi or blood-vessels, being more resistant than the lung parenchyma, may form prominent ridge- or band-like swellings in the lining walls, or may occasionally form septa crossing the cavity. In some cases the walls of either the neighbouring bronchi or blood-vessels, or both, may by this cavity formation be so weakened in places that their lumen becomes distended, forming bronchiectatic cavities and aneurisms respectively. Such aneurisms may rupture and cause serious or even fatal hæmorrhage. Less commonly the vessel wall is directly eroded, causing a less severe form of hæmorrhage. Neighbouring cavities often communicate with one another. A cavity once formed never heals up, though it is often greatly diminished in

size and contorted by the surrounding fibrosis, and it may even be completely shut off from all communication with the bronchi or the rest of the lung and thus become, and even remain, quiescent. Usually, however, the cavities remain in communication with one or more bronchi which are in a state of inflammation owing to the irritating discharges from the cavities passing through them. Various accidental germs thus reach the cavities from the inspired air, among the commonest and most notable of which are pyogenic forms. Pus is consequently formed in the walls of the cavity, and accumulating therein is coughed up at intervals, constituting the typical purulent nummular sputa of well-marked phthisis. The system is now being gradually poisoned by septic as well as by tubercular toxins. Many hold, indeed, that the chief symptoms of a typical case of established chronic phthisis are due to the septic and not to the tubercular germs, and that so long as this contamination is absent there will be some fever but few or no other symptoms. The disease may show frequent periods of quiescence of varying duration, only to break out again afresh. The extension in such cases is either by local extension, as already described, or by bronchial extension (the softened caseous contents being partly aspirated into fresh bronchi and alveoli) or by the lymphatics or blood-vessels. The primary patch thus becomes bigger and new patches are formed in other parts of the lung. The pleura is invaded ere long by an extension of a caseous or suppurating focus or cavity into it. A general pleurisy (simple, tubercular or purulent, acute or chronic) may follow, or the pleurisy may be local, particularly when chronic, and form a dense covering to a patch or a cavity which helps to protect the pleura from further invasion. The pleural surfaces (costal and parietal) may become more or less extensively adherent. When a pulmonary cavity ruptures into the pleural cavity, air escapes into it, constituting a pneumothorax. It is always a serious

complication, as it not only seriously interferes with the expansion of the lung, but is quickly followed by an empyema, since the air always contains pyogenic germs. As the patient's strength gets more and more reduced, more and more pulmonary tissue is invaded, until at last a rapid and acute extension occurs and death supervenes. The lungs in such a case, for both lungs are usually affected, though unequally, show one or more chronic lesions (*i.e.*, caseous patches or cavities surrounded by much fibrosis) and, in addition, many acute lesions (*i.e.*, recent miliary tubercles or broncho-pneumonic caseous patches, or even acute cavity formation). The bronchial glands always show caseation and pigmented fibrous changes; but the tubercular process as exhibited by them is usually small compared with that of the lung. This description applies to the form of chronic pulmonary tuberculosis most frequently met with, and often called chronic caseous or caseo-fibrous phthisis, to distinguish it from a still more chronic form in which fibrosis is far more prominent than caseation all through the disease; and hence the lung at the end is greatly shrunk and distorted, adherent by a densely thickened pleura to the chest wall, which is consequently depressed and contracted. The lung substance shows pigmented, dense, fibrous bands everywhere, continuous with the thickened bronchial walls and with the pleura, deeply congested bronchi with numerous bronchiectatic cavities, the lung substance between being scanty and emphysematous. This form is usually called fibroid phthisis.

Accompanying changes in other organs.—These are due either to the extension of the bacillus or the action of its toxins or of those of the contaminating germs upon the bodily tissues. The extension may be direct, as when the bacilli reach the larynx through the process of expectoration, or the gastro-intestinal canal through some of the sputum being swallowed (common in children),

and set up ulceration in these organs. It is usually most marked in the lower portion of the small intestine or the region of the cæcum. Another method of spread is by the blood-stream producing acute miliary tubercles in the liver, spleen, kidney and in the serous membranes, such as the pericardium and the meninges. The combined toxins cause granular, fatty and waxy degeneration, particularly in the liver, spleen, kidney and intestine. In the latter position waxy degeneration produces diarrhœa, often a distressing feature in the later stages of phthisis. The whole gastro-intestinal canal is upset in these later stages, dyspepsia being frequently a marked condition when the general nutrition also suffers. The heart muscle is degenerated, but the right heart is mostly hypertrophied, owing to the fibrosis in the lung. The heart is otherwise, or as a whole, often rather small. Chronic valvular disease may co-exist, but the pulmonary venous congestions of ordinary valvular disease are rather unfavourable to the development of tubercle in the lung, whereas chronic valvular disease (congenital) of the pulmonary valve is, on the other hand, distinctly favourable to it.

Symptoms.—In most cases the disease has been in existence for some time before the symptoms appear. These may appear suddenly, suggesting an ordinary acute lobar pneumonia, a complication, which indeed is often, if not always, due to the accompanying pneumococcus. In the great majority, however, the onset is gradual, beginning in various ways, *e.g.*, with the symptoms of a neglected cold, of indigestion, anæmia and weakness, of hæmoptysis, of laryngeal trouble. They are best studied in detail by referring them (as indeed should be done in all diseases) to the local pulmonary lesions and the general toxic effects. The most important of the former are *cough, sputa, hæmoptysis, shortness of breath, and pain in the chest*, and of the latter *fever, disordered digestion, anæmia and loss of energy*,

emaciation, sweating at night, an irritable heart and quick pulse. The *cough* is one of the earliest and most important of the symptoms. It is at first hard, short and hacking, though later it becomes softer and easier. It may occur in paroxysms, particularly at night or on getting up in the morning, when it is apt to cause vomiting. *Sputum* may be absent at first: when it appears it is for a time scanty and mucous, gradually becoming more plentiful and purulent, until, in the fully developed disease, it is expectorated in thick purulent masses, often described as nummular. It should be examined particularly for tubercle bacilli and for elastic fibres. The presence of the former clearly establishes the tubercular character of the lung mischief. Failure to find them, on the other hand, does not exclude tubercle, but when this failure occurs upon oft repeated examinations, say fifteen to twenty, it is strongly presumptive of the non-tubercular character of the disease. The presence of elastic fibres merely indicates destruction of the lung tissue, not necessarily tubercular, but suggestive thereof, in so far as tubercle is much the commonest cause of destruction of lung tissue. The number of tubercle bacilli found in the sputum is no indication of the rate of progress of the disease. *Hæmoptysis* occurs in about fifty per cent. of all cases at some stage of the disease, early and late being the two commonest stages. In the former it is usually small in amount and oft repeated, and may be due to mere diapedesis or erosion of capillaries; when larger, say a cupful or more, it is most probably due to erosion of a larger vessel. It may simply tinge the sputum or be brought up in mouthfuls of frothy or even pure blood, with or without being preceded by cough. It may be swallowed and hæmatemesis result. The larger amounts occur most frequently in the later stages of the disease, and may even be the cause of death. They are usually due to the rupture of an aneurism of a branch of the pulmonary artery. Such aneurisms, of

the size of a pea to a hazel-nut or larger, are frequent in the walls of cavities. *Shortness of breath* does not appear except in the more rapidly-advancing stages of the disease or when anæmia is pronounced. The lungs may be extensively involved without causing breathlessness, even on exertion, provided the progress of the disease be sufficiently slow. *Pain in the chest* is mostly due to the straining caused by coughing, but in some cases it is due to pleurisy. The lesions in the lung are rarely, if ever, attended by pain. Among the general disturbances, *the fever* is one of the earliest and most important. The prevailing type is a moderate afternoon or evening rise, with a fall, often below normal, in the morning. The readings should be taken frequently, every two hours in many cases, as the course of the temperature is one of the best guides throughout the disease of the progress it is making. A high temperature means active extension. Some cases show exceptionally little or no fever, but usually it is marked, slight, or absent, according as the disease is active, stationary or quiescent. The higher temperatures (say over 101°F.) are believed by some to be due to the toxins of accompanying inflammatory organisms, and not to those of the tubercle bacillus. Exercise and excitement tend to raise the temperature. *Disordered digestion*, causing loss of appetite and dyspepsia, while occurring throughout the disease, is most frequent towards the later stages. *Emaciation* is another feature, particularly of the later stages, when it is often extreme. It is greatest when the fever, the dyspepsia and the vomiting are most marked. It is sometimes, though not usually, seen in the early stages. Indeed, as already remarked, early tubercle seems rather to favour putting on of flesh, so that while gain in weight is at all times a favourable sign in recognised tubercle, it is rather in favour of a diagnosis of early tubercle than against it in doubtful cases. *Anæmia* is often present. It may become extreme at any stage of

the disease, but is usually most marked in its later stages, when there is often a hectic flush upon the cheeks. It is a secondary anæmia, attributable to the toxins of the tubercle bacillus and the accompanying germs acting both directly on the blood and by the causation of dyspepsia, sweating and vomiting. *Loss of energy.*—At first it may be merely listlessness and disinclination to exertion, but later there is weakness and prostration, which become greater as the disease progresses. *Night sweats.*—They are very common, usually occurring in the early morning. They, like the fever, indicate active disease. They are often severe and drenching, particularly after cavities are formed. *Irritable heart and quick pulse.*—The pulse is usually rapid, almost always over 100 when the disease is active, and readily increased by very slight exertion.

PHYSICAL SIGNS.—The apices of the lungs usually show the earliest signs, hence they should be examined with the greatest care, both anteriorly, above, behind and below the clavicle, and posteriorly in the supraspinous fossæ, both when the patient is standing up and lying down. The most important of these early signs are deficient expansion, dulness and fine crepitations. The last named are met with perhaps most frequently of all as the first indications of the disease, and they are always of high diagnostic importance, particularly when present in only one apex. Wavy, interrupted or cog-wheel-like inspirations are also often recognisable. The interscapular regions sometimes show these signs even before the apices, and in exceptional cases they may be present only in some part of the bases of the lungs. As the disease advances, the signs of consolidation become more marked, and bronchitic signs become prominent, particularly after softening has occurred. Diminished movement, dulness, increased vocal fremitus, bronchial breathing, increased vocal resonance and bronchophony all point to consolidation. A thickened pleura will more or less modify these signs.

In extensive fibrosis there is marked retraction and diminished expansion of the chest on the affected side, but the dulness on percussion is often much less marked and extensive than we expect, owing to the concurrent emphysema. Cavities are not usually detectable until they reach the size of a walnut, and not always even then. The most characteristic signs of a cavity are diminished expansion, increased vocal fremitus, a defective or hyperresonant percussion note, whose pitch is often increased when the mouth is opened or altered by a change of posture upon the part of the patient (owing to altered position of the fluid contents), or possessing a cracked quality (the cracked sound) when the cavity is large; bronchial, cavernous or amphoric breathing, coarse bubbling râles (except when the cavity is dry) which may have a metallic or ringing quality, particularly on coughing; bronchophony and pectoriloquy. All these points must be taken into account in making a diagnosis, as some of them, *e.g.*, the hyperresonant percussion note, the cavernous breathing, and the ringing râles, may be due to an area of consolidation bordering on a large bronchus. The effect of coughing, of change of position, and the variations from day to day, must be closely studied.

Diagnosis.—The early stages before sputa appear present the greatest difficulty. Attention has to be paid to the early signs and their gradual and spontaneous appearance. When sputa appear, the discovery of tubercle bacilli puts all doubt at rest. The temperature chart must be carefully studied, and may suggest tubercle before any other indication does. In doubtful cases the tuberculin test (a small dose) may be employed.

Prognosis.—Some cases last only a few months, others live for many years. Social position has much to do

with the result, for generally the more favourable it is, the slower is the course of the disease. Its average duration has undoubtedly greatly increased during the last twenty years. There has been a steady improvement, and this has been most marked of recent years, owing to the more enlightened methods of treatment. It is difficult to give reliable figures, but the suggestion may be hazarded that among the poor and least favourable cases the average duration is not less than three years; while among the better classes it is at least as high as seven years. The temperature and the digestive powers are the best guides to the progress. Complete recovery undoubtedly occurs, and it is the more likely the earlier the case comes under treatment. Quiescence and recrudescence are still more common, so that all cases should remain under supervision for long periods after the disappearance of all signs of activity, and it may be that many such recrudescences will be avoided and complete cures follow.

Treatment.—Prophylaxis.—We know how tubercle is apt to be propagated by air, milk, meat, &c., and therefore we should specially guard persons hereditarily predisposed or delicate from the risk of infection. Tuberculous cows should be removed from dairies, and no tuberculous meat allowed to find its way into the market. The reader is referred to page 121, where remarks on the prophylaxis of general tuberculosis have already been made; but in connection with chronic pulmonary tuberculosis there are some special points requiring attention. The disposal of tuberculous patients in order to safeguard the public is most important. If very ill and, as is generally the case, also very infectious, such individuals should be treated in hospitals where only cases of a similar nature are received, unless it is possible to have them nursed and treated at home under conditions which do not endanger

the lives of others. For early, or what we may call curable cases, the open-air plan of treatment in sanatoria is desirable, because there should be as much separation as possible between the consumptive and the healthy, and still more between the consumptive and those who have a hereditary tendency to the disease. From the point of view of the patient, the sanatorium will be referred to later.

There is no more important point than the destruction of infectious sputum. The patient must spit into a wide-mouthed vessel which he can carry about with him, and which can be thoroughly cleansed after the contents are destroyed. A little antiseptic lotion, such as carbolic acid or lysol, should be kept in this sputum bottle so as to prevent the possibility of any of the sputum drying and so permitting tubercle bacilli to become diffused throughout the air. The patient must be prohibited from spitting on the floor of rooms, in street cars, railway carriages or even on the pavement, although it is true that sunlight and fresh air do, in time, destroy the organisms. Handkerchiefs should never be used for purposes of expectoration; and where, from great weakness, the patient is unable to spit into a proper sputum dish, rags or Japanese paper handkerchiefs should be employed and immediately after use should be burnt. Tubercular patients must sleep alone, and if possible in a separate room. Relatives must be enjoined to avoid kissing them on the mouth, and in the case of men the moustache and beard should either be shaven off, or if for some reason that is undesirable the moustache and beard should be regularly cleansed with an antiseptic lotion, and this should be invariably done after much expectoration has occurred.

The rooms inhabited by tuberculous patients should be dusted with a damp duster and the floor swept after a free use of wet tea leaves, and it is infinitely better to have bare boards and no carpets. Fresh air and as much sunlight as possible are also essential.

The life of predisposed persons should be carefully arranged so as to permit of plenty of exercise in the fresh air. The work of such an individual should be as much in the open air, and as free from dust and overheated and badly-ventilated workrooms as possible. The question of marriage is a very important one; it is highly undesirable for two predisposed individuals to marry, even although neither of them may have any active disease, but it is usually difficult to exercise much control in these matters. Persons the subjects of active disease should be prohibited from marrying altogether, and more especially in the case of women; the risks which a female once a tuberculous patient, even though cured, may run by entering on the married state cannot always be accurately gauged. There is the risk that after child-birth the disease may make rapid strides, and the children may be, and very often are, predisposed to the disease.

General Treatment.—Tuberculous patients should be nursed in large airy rooms with plenty of sunlight and wide open windows: indeed, if possible, they should live almost completely in the open air. The cold, damp, inclement weather of the greater part of the British Isles during at all events a portion of the year is not suitable for tuberculous patients, and where able to afford it they should be advised to go to a high and dry, even although cold place of abode during at all events the four months of winter. Davos, Clavadel and Colorado Springs are good types of high mountain health resorts (5000 feet and over). At a lower elevation may be mentioned many places above Montreux, such as Caux and Chateau D'Oex (3000 to 4000 feet). These latter places are bright and sunny in winter, and are free from excessive winds. High altitudes do not suit every case, and especially those with rapidly progressive disease, cases with much hæmorrhage or with serious laryngeal involvement, cases who have

much emphysema, and those whose hearts or kidneys are affected. For those patients who are able to afford it, and who do not suit a high altitude, the warm sunny south is to be commended, and Egypt, the Riviera, and Algiers are amongst the most suitable places, because, even in cases which are almost hopeless, life is rendered easier and more endurable. South Africa is in many parts extremely suitable, especially as the land rises quickly soon after the sea is left; but dust storms are not uncommon, and the places to which patients are sent should be carefully selected. A sea voyage for an early case is admirable treatment. There are, however, some favoured spots in the South of England possessing a maximum of sunshine in the winter, and they have the great advantage of being near home. Where a patient is dying he should on no account be sent away, because, where there is little hope of cure, he exchanges the comforts of home for much that is unsatisfactory abroad, and, further, it is eminently desirable that infectious persons should be prevented from endangering the health of their fellow-men.

The principles of general treatment are very simple. The patient should be hardened to stand cold—not too suddenly but by degrees—and this the open-air life does most effectively. An open-air shelter placed in a sunny position, and so arranged that the patient is protected from the prevailing winds, should constitute the patient's sitting room. The chest should be sponged with cold water every morning and the clothing should be carefully regulated. There must be no excess, although he should wear wool next the skin both night and day, and the number of blankets used at night, while sufficient to keep him warm, must not tend to promote sweating.

A full dietary is necessary, and many authorities recommend a considerable quantity of raw meat every day. This can be easily given in the form of sandwiches, which can be made quite palatable. Milk, soup, fish, &c., and all

nourishing kinds of food may be freely ordered, but it is questionable whether forced feeding is good in most cases. Cream is admirable, although it is of doubtful value in cases in which waxy changes are present. Cod-liver oil is in reality a food, and it may be given pure or as an emulsion. The oil alone has certainly to some patients a peculiarly nauseating taste, but this may be concealed by a little bit of dry bread, a few drops of lemon or orange juice, or a bitter tonic, such as quassia or calumba taken immediately after the oil. Begin with a teaspoonful of the oil and increase to a tablespoonful once or twice a day, and give it *with* the food or, as some physicians prefer, at bedtime. Jaccoud ordered it in enormous doses, but cream and butter are more palatable fats and probably quite as good. One objection to cod-liver oil in excess is the tendency to the taste coming back into the mouth, after it has been swallowed, owing to eructations. On this account, children, and sometimes adults, are rubbed with cod-liver oil, but this renders the patient horribly odoriferous and is by no means so beneficial.

Recommend gentle exercise, the amount depending on the severity of the case, and at the Alpine health resorts the kinds of exercise permitted must vary. Some patients can skate, ski, toboggan and enter into all the life of the place without harm, while others must be content with walking exercise. Dancing in hot rooms should be forbidden, and excitement for all serious cases is extremely prejudicial. In many instances carefully graduated pulmonary gymnastics are good, but in cases where there is extensive disease they must be carried out under medical supervision; hæmorrhage, however trifling, indicates that they are harmful. There seems no question that in summer, when many Alpine sanatoria are unsuitable or are closed, glacier walking, for which we have pled elsewhere, is of great value if the patient is able to walk and climb in moderation.

Medicinal Treatment.—Can we attack the tubercle bacillus by any known remedy? Guaiacol (15 to 25 minims in capsules), guaiacol carbonate (10 to 20 grains in cachets), creosote, iodoform (2 to 3 grains in pill) have, amongst other remedies, been administered internally in the hope that they might exert a beneficial effect upon the disease. Certainly turpentine taken in capsules exerts an antiseptic influence during its excretion by the lung. Inhalations of antiseptics vaporised or in the form of spray have been recommended, and amongst the remedies usually employed in this way are creosote, guaiacol, essential oil of cinnamon, formol, menthol, and others. A useful form of administration in certain cases is by intra-tracheal injection, and the following prescription may be employed—guaiacol 2 parts, menthol 10 parts, sterilised olive oil 88 parts, one drachm being injected thrice daily by means of the intra-tracheal syringe. The objection to this method is the difficulty of making certain that much or any of the injection will really reach the damaged lung.

Certain preparations of arsenic, and in particular the cacodylate of soda, of which the dose administered represents an enormous amount of arsenious acid, obtained at one time much notoriety; but, according to Sir Thomas Fraser, the cacodylate holds its arsenic in a form which renders it absolutely inert. We need not discuss the multitudinous remedies of bygone days, such as goat's blood, &c., but it is necessary to refer to tuberculin, although that too has been mentioned already on page 122 in connection with the treatment of tuberculosis generally. Since that page was written, Wright's work has established the value of tuberculin T.R. as an important method of treatment. It is true that the method is of most value in cases of tubercular disease of a very chronic nature, and particularly involving glands, skin, bones and joints. The method is certainly not so applicable to the lungs, but as it has not previously been described, reference to it must be made here.

In the first place, the opsonic index of the patient's leucocytes must be obtained ; or, in other words, the number of tubercle bacilli which each leucocyte can eat up or take into its own body. This power is largely due, not to the leucocyte in itself, but to the blood-serum of the patient. Patients suffering from chronic tuberculosis generally have what is called a low opsonic index, and it is desirable to have a standard from a normal non-tuberculous individual which we may call unity. In the kind of cases just mentioned the index may average only .5 or .4, whereas in acute cases of tuberculosis the index may reach 1.5, 2, or even more. Following an injection of tuberculin T.R. of about $\frac{1}{3000}$ th of a milligramme, the opsonic index falls, and only after several days does it rise, probably to a higher figure than it was before. The opsonic index represents the protecting power of the patient's leucocytes (but by means of his blood-serum) from the attack of the tubercle bacillus, and therefore it is necessary to abstain from the administration of a second dose of tuberculin until this negative phase has been replaced by the positive. The technique of the procedure cannot be fully described here, but it has been found sufficient to use dead tubercle bacilli in studying the numbers which each leucocyte can take in. The leucocytes need not be those belonging to the patient, but it is essential that the blood-serum should be obtained from the patient under examination. The leucocytes are washed with saline solution, and when mixed with blood-serum of the patient and dead tubercle bacilli the mixture is incubated in a given time and the number of bacilli in each leucocyte averaged. The procedure by which the opsonic index is obtained implies a considerable amount of hard work, especially as the blood of the patient must be examined frequently. Great improvement follows the raising of an abnormally low opsonic index, although, as already stated, pulmonary tuberculosis cannot be treated so successfully as cases of the disease

in a chronic form in the other parts of the body already mentioned.

Treatment of Special Symptoms.—In describing the treatment of special symptoms we shall follow the order taken up under the clinical features. *Cough* is often soothed by the use of remedies similar to those suggested for bronchitis on page 506. An alkaline cough mixture such as sodium bicarbonate (10 grains), ipecacuanha wine (10 to 20 minims) with syrup of tolu, and infusion of senega helps to render viscid sputum more free; while should the sputum become excessive the spirits of chloroform, of ether and of ammonia are more applicable. When the coughing is persistent, liquorice lozenges, a linseed poultice, gargling the throat, and sometimes inhalations of creosote, menthol, &c., are beneficial. Often the cough prevents sleep, and then a mixture such as liquor morphinæ hydrochloratis (10 minims), dilute nitric acid (10 minims), dilute hydrocyanic acid (3 minims), with syrup of tolu and acid infusion of roses is found to be soothing. When vomiting is induced, the combination of dilute nitric acid, ipecacuanha wine, and infusion of quassia may be tried, or small pieces of ice given to suck.

If the *sputum* becomes foetid, then inhalations of guaiacol, creosote, menthol, or essential oil of cinnamon should be accorded a lengthy trial. *Hæmoptysis*, when excessive, calls for absolute rest in bed, freedom from all excitement, and the use of morphia, such as the prescription given on page 531, which contains that drug in combination with dilute sulphuric acid. Low diet, saline purgatives, and sometimes the use of ice, both internally and applied externally, are often advisable. Where the hæmorrhage is continuous and alarming, bleeding from the arm may be considered, but it is desirable first to try adrenalin and also calcium chloride so warmly recommended by Wright. Turpentine in capsules acts as a hæmostatic as well as an antiseptic. Make the patient lie on the affected side in order to prevent the blood

from entering the other lung. *Shortness of breath* may be treated by alcohol, or the aromatic spirit of ammonia, spirit of chloroform, and spirit of ether, in a mixture containing 20 minims of each. *Pain in the chest* of pleuritic origin should be treated by poultices, blisters, or in very chronic cases by iodine. *Pyrexia* requires rest, and, if excessive, cold sponging, and often quinine. *Disorders of digestion* should be treated by acid tonics combined with tincture of nux vomica and a varied dietary. Attend to the bowels when necessary and check diarrhoea by suitable remedies. *Emaciation* implies drain on the system by the febrile process and by the disease, and in attempting to treat the condition the digestion must be improved if possible, and cream, nourishing foods, and so forth ordered. *Anæmia* should be treated by iron, and especially the proto-salts; sometimes arsenic in small doses is added. *Loss of energy* calls for tonics, nourishing food, and alcohol. *Night sweats* may be largely obviated by sponging the patient so as to prevent the nightly rise of temperature. Atropin sulphate in doses of $\frac{1}{100}$ th grain, quinine in doses of 5 grains, and camphoric acid in doses of 20 grains, are a few of the many remedies which have been found applicable in checking sweating. It is important to remember that after sweating the nightdress requires changing, and, as in all such cases, that garment should be made of thin flannel.

One complication, besides hæmoptysis, which demands a word of description is *pneumothorax*. The consequent pain and dyspnoea are often urgent, and opium in some form may be necessary. If the pressure rises very high, tapping with a fine needle may be requisite, and local blisters and counter-irritation are of value.

SYPHILITIC PNEUMONIA.

Forms.—Syphilitic disease of all forms is rare in the lung. The commonest is the congenital diffuse interstitial pneumonia, the next is the gumma, and the least common is the acquired diffuse interstitial pneumonia. None of them is of much clinical importance. The congenital diffuse interstitial pneumonia occurs occasionally in infants who are, in most instances, still-born. It is usually bilateral, and may affect the lungs in patches, or over a large area. The affected part is somewhat enlarged, firm and solid and of a greyish-white colour, hence the name "white pneumonia" given to it long ago. Microscopically, it shows great thickening of the interalveolar septa by a young fibrous tissue growth, thickening of the walls of the blood-vessels, and compression of the alveoli, whose lining endothelium may be swollen and cubical in shape, or proliferated and desquamated, so as to completely fill the cavity. This cellular exudate may undergo fatty degeneration. In all cases the affected part of the lung soon becomes airless.

The GUMMA may be congenital or acquired. In the former case it may be present along with the diffuse interstitial pneumonia just described, or without it. The number varies, but several are usually present. They vary in size from a pea to a walnut or larger, and have the usual greyish-yellow centre surrounded by a capsule of fibrous tissue. They are most commonly situated near the root, and not so often beneath the pleura or elsewhere in the lung. They may ulcerate into a bronchus and the softened centre be expectorated, forming a cavity as in tubercle. The acquired diffuse interstitial pneumonia is seen most frequently near the root, extending outwards, or, less commonly, near the pleura, extending inwards, as firm fibrous bands specially surrounding the blood-vessels and the bronchi.

Treatment.—The congenital “white pneumonia” is almost always found in still-born children. Gummatous affections of the lungs are rarely diagnosed, but where the condition is suspected, potassium iodide should be administered in 10 grain doses (for adult patients) thrice daily, and gradually increased until double or treble that dose is being taken.

ACTINOMYCOTIC PNEUMONIA.

Actinomycosis occurs in the lung not infrequently. It may be an extension from the mediastinum or the bronchi, or occur primarily in the lung in the form of whitish nodules surrounded by congested zones. The nodules soften and are discharged as purulent sputa, leaving cavities surrounded by much fibrous tissue. This purulent softening of the pulmonary nodules is a characteristic feature. It occurs more quickly in pulmonary than in other forms of actinomycosis, and enables the true nature of the pneumonia to be discovered through the presence of the actinomyces in the sputa.

Treatment.—Potassium iodide is believed to have an almost specific action in certain cases of the disease, and is certainly worth a trial, otherwise treatment must be on general lines and is not hopeful.

TUMORS OF THE LUNG.

Simple tumors, *e.g.*, lipoma, chondroma, fibroma, &c., are so rare that they do not need further notice. Malignant tumors, on the other hand, particularly secondary ones, are common. Both cancers and sarcomas occur as primary

growths, the former originating in the mucous glands of the bronchi and the latter in the connective tissue or endothelium of the alveoli (endothelioma). Primary cancers usually radiate into the lung along the sheaths of the bronchi. They are usually one-sided, at least for a time, while secondary tumors are almost always bilateral and multiple. Primary cancer of the lung rarely gives rise to secondary growths in other organs.

Symptoms.—They depend upon the size and mode of growth of the tumors. There is usually pain from involvement of the nerves at the root of the lung or the pleura, cough from affection of the bronchi, sputa (sometimes of a red-currant jelly or prune-juice colour), hæmoptysis, dyspnœa (often very severe), anæmia and general weakness. When the growth compresses a bronchus, the part of the lung supplied by it may become atelectatic and subsequently pneumonic. The physical signs are those of bronchitis and consolidation.

Treatment.—The treatment is mainly symptomatic. Paracentesis has to be carried out where a large pleural effusion gathers. The patient is apt to be alarmed by the constantly recurring hæmoptysis, which is, however, not generally large in amount. Give dilute sulphuric acid and try adrenalin and other styptic remedies. Unfortunately they are not likely to do more than temporarily check the hæmorrhage.

PARASITES OF THE LUNG.

A considerable number of parasites may be found in the lungs of man, but the only one of any practical importance clinically is the *Tænia Echinococcus*, constituting hydatid

disease of the lungs. It is rare in this country, though more common (about three times as frequent) in Australia. It may be primary, when the ova are probably inhaled, or secondary (much more common), when it usually spreads from the liver. It may extend directly through the diaphragm or travel by the inferior vena cava. The hydatid cysts may be single or multiple, of small or large size. They show their characteristic contents and walls, except that the outer fibrous coat, formed by reactive inflammation in the lung, is less dense than in other organs, hence the cysts often rupture (in about half the recorded cases) into some neighbouring tissue, *e.g.*, the pleura, the pericardium, or a bronchus, when the cyst may be largely evacuated and dry up, or it may become contaminated with pyogenic cocci and suppurate. In other cases it may enlarge and cause trouble by pressure, or it may shrivel and dry up.

Symptoms.—There are usually none till the cyst ruptures, after which they will vary with the part into which the rupture has occurred. The most common are cough with expectoration of mucus or watery fluid, tinged or plentifully mixed with blood. Daughter cysts or parts of the ectocyst may be discovered in the sputa, and hooklets also on microscopic examination. Pain is present when the pleura is involved, but there is usually only slight dyspnoea, except when rupture has just occurred into a bronchus. Hæmoptysis is also apt to occur at the same time. Prior to rupture the general health usually remains good.

The PHYSICAL SIGNS indicate the presence of a solid, or sometimes of a fluctuating, swelling.

Diagnosis.—It is often very difficult. The situation and form of the area of dulness, the presence of fluctuation, the good general health, and the presence of cysts or hooklets in the sputa are the best guides.

Prognosis.—Hydatids of the lung may exist for years without much affecting the general health, but spontaneous rupture may cause death by flooding the lungs, the pleura, the pericardium, or from hæmorrhage or pneumonia. Abscess or gangrene may supervene. Artificial evacuation, on the other hand, by surgical operation offers a much better outlook.

Treatment.—The treatment should be directed to an attempt at tapping the hydatid cyst or cysts. As indicated above, evacuation by incision may be necessary, and should be at once performed where suppuration has occurred, in preference to running the risk of gangrene by any undue delay.

V.—DISEASES OF THE PLEURA.

These may be grouped under the following headings:—Circulatory Disturbances, Inflammations, Tumors and Parasites.

CIRCULATORY DISTURBANCES.

HYDROTHORAX.

Hydrothorax, or dropsy of the pleural cavity, is most frequently met with as a part of the general dropsy of chronic kidney or chronic heart disease. It is generally bilateral, though not always equal in amount on the two sides, the fluid being clear and watery. It may be right-sided only, as in pressure of the azygos veins by an intra-thoracic tumor or aneurism. The lung or lungs are correspondingly collapsed.

Symptoms.—Shortness of breath is generally the only noticeable symptom, and even it may be absent when the fluid collects very slowly. Usually the symptoms of the primary disease overshadow those, if any, due to the hydrothorax.

Treatment.—The treatment of most cases of hydrothorax is two-fold. *First* try to help Nature to remove the effusion by purgatives such as Henry's solution, by diuretics such as diuretin or the spirit of nitrous ether, and by tapping when necessary; and *second*, treat the heart or lung conditions which are primarily responsible.

HÆMOTHORAX.

Hæmothorax, or blood in quantity in the pleural cavity, is caused by a fractured rib tearing an intercostal artery or the lung, or by the rupture of an intra-thoracic aneurism, occasionally by cancer or tubercle of pleura or lung or by some blood disease, such as leukæmia or scurvy. The blood may clot or remain fluid. It is usually rapidly absorbed when the pleura is healthy. In other cases it may be contaminated by pyogenic or other germs.

Symptoms.—They are much the same as in hydrothorax, except that there may be syncope when the escape of blood is sudden and severe.

Treatment.—The treatment depends much on the cause of the hæmorrhage. Ergot and styptics are useful in certain cases; in traumatic hæmothorax, as from fractured rib, absolute rest in bed, often with a hypodermic injection of morphia, should be ordered.

CHYLOTHORAX.

Chyllothorax, or chylous fluid in the pleural cavity, is rare and follows upon rupture of the thoracic duct. It can only be recognised after exploratory puncture of the chest wall.

PNEUMOTHORAX.

Pneumothorax, or air in the pleural cavity, may be included here as it is a disturbance in the air circulation of the lungs. The air may enter from without, by puncture of the chest wall (rare), or from within, coming from the lung, œsophagus or stomach (gastric ulcer). The lung is its most common source, and the most common lesion thereof which leads to it is tubercle. A cavity (most frequently a small quickly-formed one) or caseous area in the lung may penetrate the pleura. Abscess, gangrene, or even emphysema (rare) of the lung may act in the same way. In some few cases a pneumothorax may be produced by the entrance into the pleura of some gas-forming bacillus, e.g., the bacillus lactis aerogenes or bacillus coli. In these last cases the gas is mainly CO_2 , CH_4 , or N ; but in the previous forms, where air enters, it soon comes to consist of nitrogen mainly, with some CO_2 , the oxygen having been largely absorbed. The gas may be present under very considerable pressure, so that it escapes with a hissing sound on puncture of the chest wall and with sufficient force to blow out a candle. In such cases the lung will be collapsed much the same as in hydrothorax. Owing to the air being usually contaminated with pyogenic organisms, a pleurisy of a serous or purulent character (pyopneumothorax) soon results.

Symptoms.—They vary according to whether the pneumothorax occurs in a person with healthy or with slightly affected lungs or with much affected lungs. In

the former case there is usually sudden and severe pain in the chest after some exertion, breathlessness and cough (without sputa), small frequent pulse, and pallor or cyanosis. In the latter case there may be no special symptoms until a pyopneumothorax appears, when the cough may be accompanied by purulent sputa, and the patient will be found to lie most comfortably on the affected side.

The PHYSICAL SIGNS are often very characteristic, viz., diminished expansion on the affected side, loss of vocal fremitus, tympanitic percussion note, and absence of breath sounds. Vocal resonance is generally lost, but sometimes there is bronchophony or pectoriloquy. Dulness on percussion over the lower part of the chest, with a splashing sound on succussion, indicates a pyopneumothorax.

Prognosis.—When the lungs are healthy, the prognosis is very good, recovery following in one to six weeks; when the lungs are diseased the prognosis is always grave. In such cases the pneumothorax generally shortens life, but the longer it lasts the less influence it seems to have, except when pus supervenes, when a rapid downward course is usually the result.

Treatment.—In a traumatic case, as from fracture of a rib, strapping the chest and the application of a bandage may be tried. Pain may call for the application of poultices, and where the pressure rises very high we may need to tap, an operation which should be deferred as long as possible, because the air acts as an excellent splint to the wounded lung, and if not in excessive and increasing amount it is readily absorbed. Some surgeons have recommended introducing nitrogen gas or sterilised air in place of the air already in the sac, but this seems superfluous. Stimulants may have to be administered

and careful dieting is called for. When pain and restlessness are excessive, opiates are indicated.

Many cases of pneumothorax are due to tubercular cavities, and here the treatment is less hopeful. Should the patient be much collapsed, stimulants may be necessary. Strapping and bandaging, poultices, and a hypodermic of morphia may be required, depending on the nature of the case and the extent of the pneumothorax. Most phthisical cases of pneumothorax tend to become pyopneumothorax from the presence of pus-producing organisms, and then the treatment suitable for that condition must be carried out.

INFLAMMATIONS OF THE PLEURA— PLEURITIS OR PLEURISY.

ACUTE PLEURISY.

Etiology.—Like pericarditis, it is a secondary lesion most frequently. It may arise by direct extension of the inflammation from some neighbouring organ, or indirectly by the blood-stream. The extension may be from some thoracic organ or structure such as the lung, the pericardium, the mediastinum, the vertebræ, the ribs, or from the abdomen through the diaphragm, as in hepatic, supra-hepatic, or splenic abscess, or in gastric ulcer. In the second group, where it originates indirectly, there may be a lesion in some distant part of the body in the form of an inflammation (Bright's disease), a suppuration (pyæmia), an ulcer, or a necrosis (typhoid fever). The disease may be a general systemic one, as in the ordinary *acutè* infections. In all such secondary pleurisies the bacterium causing the pleurisy is generally the causal organism of the primary disease, but not necessarily so, as other germs may enter the body (notably those of ordinary inflammation and suppuration) during the course of these diseases and cause

the pleurisy. This may be the case even when the primary disease is in the lung, where it is often taken for granted that the same bacterium as has caused the pneumonia will necessarily also be the cause of the pleurisy, *e.g.*, if the pneumonia (the primary disease) be tubercular it is concluded that the pleurisy is also tubercular. This is not always the case, as the pleurisy may be caused not by the tubercle bacillus itself but by some complicating or contaminating organism. The other great group of pleurisies—those of primary origin—while less frequent than the secondary, is more interesting clinically. Primary pleurisies are met with after chills, or in the course of certain general diseases of non-bacterial origin, or arising in healthy persons without any discoverable preceding condition which might be regarded as having a predisposing influence. They may be caused by either a non-specific or specific organism. In the latter group the tubercle bacillus is certainly of most frequent occurrence, but the statement that it is the most frequent of all the causal germs is open to question. It is mainly based upon clinical experience, which shows that many primary pleurisies end in tuberculosis, but it is not borne out to the same extent by the evidence obtained by bacteriological examination during life and after death. That a certain proportion, and that a fairly large one, is tubercular is most probably true, but there is at least a considerable proportion caused by other germs. Of those belonging to the specific group, certain cases have been attributed to the influenza bacillus, though on somewhat uncertain grounds, and others to the typhoid bacillus; whilst, of the non-specific germs, the diplococcus lanceolatus of Fränkel is certainly the most frequent. It is probably as common a cause of primary pleurisy as is the tubercle bacillus, or nearly so. Other germs of less frequent occurrence are the streptococcus, the staphylococcus, and the bacillus coli.

Morbid Anatomy.—The changes are those characteristic of inflammation of any serous membrane, viz., redness, loss of gloss, and exudation. The description of the morbid changes given under pericarditis apply equally well here, and need not be given in further detail. The character of the exudate is used here also as in pericarditis to distinguish pleurisies into varieties: thus we speak of serous, sero-fibrinous, fibrinous or dry, purulent, and hæmorrhagic pleurisies. In the *serous variety* the fluid is clear and watery, and does not deposit fibrin after its withdrawal from the body. Most serous pleurisies have a certain amount of fibrin present in the form of threads, flakes or masses, and hence the term *serofibrinous* may be said to include all serous varieties. There is generally some fibrin on the surface of the pleura and some floating free in the fluid or deposited from it after its withdrawal from the body. The amount of the fluid varies, and may be as much as several quarts. It may occupy the whole pleural cavity, or be loculated when it is confined by adhesions to a part of that cavity. It is highly albuminous and contains certain cells, viz., altered and degenerated endothelial cells, polymorphonuclear leucocytes, lymphocytes, and occasionally a few red blood-cells. It is generally held that a high percentage of lymphocytes or of red blood-cells indicates that the pleurisy is probably of a tubercular origin. This is not altogether a safe guide, as early tubercular pleurisies may show a high percentage of polymorphonuclears, and late pleurisies, whether tubercular or not, may show a great predominance of lymphocytes. Bacteriological examination of the fluid is difficult when any fibrin is present, but taking the two germs which are the commonest causes of all pleurisies, viz., tubercle bacillus and the pneumococcus, it shows that the former is far more frequently present in such serous pleurisies than the latter. In the *fibrinous or dry variety* the fibrinous material forms the chief constituent of the exudate, and hence both pleural surfaces show the

typical rough honeycombed appearances. The bacteriological examination of such pleurisies shows a great predominance of the pneumococcus over the tubercle bacillus and over other causal germs as well. In the *purulent variety or empyema*, most frequent in children, the fluid is composed of pure pus or sero-pus. The latter is well seen in those cases in which an original serous pleurisy becomes purulent as the result of imperfect aseptic precautions in tapping. The bacteriological findings in purulent pleurisies present considerable differences, and it is doubtful if any germ is predominant. The pneumococcus and the streptococcus are perhaps the most frequent, but the tubercle bacillus and others as well are not infrequent, either alone or together. The pus if allowed to accumulate most commonly burrows through the lung into a bronchus and escapes by the mouth. Pneumothorax may follow. Much less commonly it makes its way through the chest wall and is discharged externally. The commonest site of rupture in such cases is anteriorly in one of the upper intercostal spaces. In exceptional cases the pus may cease to increase spontaneously, when it gradually becomes dried up and ultimately forms a cheesy or calcareous mass. In the *hæmorrhagic variety* the exudate is largely mixed with blood. It is particularly liable to occur in tubercular or cancerous pleurisies, or in the course of purpura or other blood diseases.

Associated lesions.—The inflammation may extend to contiguous or distant organs, and thus a pericarditis, peritonitis, meningitis, &c., may arise. The most prominent direct effect is upon the lung, when the fluid in the pleural sac is considerable. The lung is collapsed in part or in whole, incompletely or completely, according to the position and amount of the pleural fluid. When the whole lung is collapsed it is bunched up as it were towards its root, being driven inwards, backwards and upwards towards its fixed part (its root), and when such

a collapse is complete the lung forms a flat airless mass lying along the vertebral column, its size being reduced to a mere fraction of the normal. Such a lung is capable of subsequent expansion for a long period of time, but if it remains collapsed too long it will not afterwards expand, and if the fluid be subsequently got rid of, the chest wall must fall in, the vertebral column be twisted, and the thoracic organs displaced so as to fill up the space which should be filled up by the lung. The resulting chest deformity is often very great, particularly in children, whose bones are more pliable than those of adults.

Symptoms.—The leading symptoms are pain, cough, shortness of breath, and fever. The pain is generally the first thing complained of. It is mostly sharp and usually spoken of as a stitch in the side of the chest. It is increased on taking a deep breath, hence the breathing is shallower and more rapid than normal. Cough is not a prominent symptom. It is irritating and dry and gains attention not so much on its own account as of the pain to which it gives rise, hence it is suppressed as much as possible. The fever varies, the usual temperatures being between 100°F. and 104°F. The pulse is correspondingly frequent, and the other usual symptoms of fever are seen, viz., anorexia, thirst, &c. The patient is unable to lie on the affected side. These symptoms may wear off and the patient be restored to health without the pleurisy altering its dry character: more commonly, however, they change owing to the onset of a serous effusion into the pleural cavity. The two layers of the inflamed pleura no longer rub against one another and the pain disappears. The cough is no longer painful. The breathing continues to be shallow and becomes more rapid, but there is now a feeling of weight on the affected side, inducing the patient to keep it undermost, so that he no longer lies on his sound

side but towards, though not actually on, his affected side. The fever usually subsides somewhat, being usually lower than in the earlier stages. In favourable cases the symptoms gradually subside until a normal condition is again reached ; but a continuance of the symptoms, particularly of a high evening temperature with morning remissions, points to the fluid becoming purulent. In certain slowly evolving cases all symptoms may be absent.

PHYSICAL SIGNS.—In the pre-effusion or dry stage there is diminished movement, friction fremitus, and friction sounds. In the later or effusion stage there is diminished movements, no friction or vocal fremitus, a wooden dulness on percussion, loss of the respiratory murmur and of vocal resonance. Later, with the gradual absorption of the fluid, there is a gradual return of the respiratory sounds and of the friction and vocal fremitus, and a disappearance of the dulness, usually from above downwards. An ægophonic or bleeting quality of the vocal resonance may be present at any time towards the upper limit of the fluid when the latter is present as a thin layer only. During the period of effusion the apex of the heart may be displaced laterally and the abdominal organs downwards. The course of the disease and the temperature may suggest that the fluid has become purulent, but the exploring needle is the only sure test of this.

Diagnosis.—Pleurodynia is distinguished from pleurisy by the absence of friction and of fever, from a thickened pleura, and from a pneumonia, &c., by the use of the exploring needle, and by a careful consideration of the symptoms and physical signs.

Prognosis.—Recovery is common. It may be complete or incomplete, leaving a thickened pleura or a damaged lung. Death from pleurisy is rare and is caused by cardiac failure. Pulmonary tuberculosis follows within a few years

in a certain number of primary acute pleurisies, particularly of the serous variety.

Treatment.—The treatment may be divided into that suitable for the different stages of pleurisy, and, lastly, reference will be made to the treatment of empyema.

The *dry stage* of pleurisy demands special attention on account of the pain. Strap the chest and bandage it, but, if the pain is very severe, poultices or, better still, one or two fly blisters (2 inches square) should be applied. A mustard leaf or poultice, iodine, and leeching are other remedies, but the fly blister is the best. A saline purge is also useful, and the patient should be placed on milk or light diet if there is much fever.

For the *effusion stage* much can be done by purgatives, diuretics and diaphoretics to help in the removal of the fluid; external counter-irritation by some of the methods mentioned above is also serviceable. Do not delay too long before tapping, because the lungs may not completely re-expand after they have been compressed for more than a limited time. It is not necessary to remove the whole of the fluid, but only a comparatively small amount, and to allow Nature to remove the rest: counter-irritation after tapping is doubly efficacious.

Paracentesis.—Use by preference an aspirator such as Potain's. The best sites are the lower interspaces, either posteriorly below the scapula or in the axillary region. The seventh or eighth interspaces are often selected, and the point should be below the upper limit of the effusion.

Carefully sterilise the needle and push it in (pulling on the skin in order to make the track as oblique or valve-like as possible) at the middle of an interspace, or just above the lower rib, so as to avoid piercing the intercostal artery. Push boldly downwards and inwards until the pleural sac is reached, when the resistance is suddenly overcome and the needle readily passes onwards. The skin may be anaesthetised

with an ether or chloride of ethyl spray for a minute or two before the operation, or cocaine may be used, and the arm of the side to be tapped should be stretched over towards the opposite shoulder, while a deep breath, taken just before the puncture is made, widens still further the interspace and prevents the patient jumping violently and possibly bending or breaking the needle. Coughing during the process of draining away the fluid indicates that the cannula is pressing against the re-expanding lung, and care should be taken to avoid causing much cough; as a rule, 50 to 60 ounces is a sufficient amount to remove from one side at a time. When withdrawing the cannula, seal up the puncture with wool and collodion, and take special care during the tapping to prevent any chance of infection of the pleural sac by its direct connection with the outer air. The dangers of paracentesis are *pneumothorax* (generally from rupture of lung with the stress of violent coughing, rarely as the result of puncture), the *expectoration of albuminous sputum*, associated with a serious œdema of the lung (but this is very rare), and *syncope* from too suddenly taking off the pressure upon the heart. Always after tapping apply a broad bandage, and it is wise to give a little alcohol immediately after, if not during, the operation. Counter-irritation after tapping is often of great advantage in helping the absorption of the remaining effusion, and pulmonary gymnastics should be systematically encouraged.

Empyema may sometimes be treated by tapping, but this is rarely sufficient, and it is generally necessary to resect one or more ribs and treat as an abscess cavity with irrigation. Many attempts have been made to drain in such a way as to try to create the nearest approach to a vacuum, and the use of a head of water placed above the patient, valve-shaped drainage tubes and other measures have been devised to try to induce the collapsed lung to re-expand.

Great care must be taken when administering an anæsthetic to the patient at the operation, and on no account must he be rolled over on to the healthy side, or sudden and fatal syncope may result.

CHRONIC PLEURISY.

Etiology.—It may follow acute pleurisy, or arise independently from the same causes as the acute form.

Morbid Anatomy.—The pleura, particularly the visceral layer, is fibrously thickened, sometimes greatly so, over the whole lung or in one or more patches. This thickening may or may not invade the lung. When it does, the interlobular septa are thickened, and a varying amount of pleurogenic interstitial pneumonia results.

Symptoms.—They may be absent, or there may be cough and some shortness of breath. The affected side of the chest is usually seen to be retracted. There is diminished vocal fremitus and percussion resonance. Friction sounds are often present, and the respiratory sounds are often feeble.

Treatment.—Probably little treatment is possible, but much may be done to raise the standard of the general health and so to guard against the attack of the tubercle bacillus. Sometimes pulmonary gymnastics are beneficial, and where friction is present, counter-irritation should be energetically carried out.

TUMORS OF THE PLEURA.

Simple tumors occur, but they are not clinically important. Primary sarcomas occur and may be of the

round or spindle-celled variety or of the endotheliomatous type. The latter is the most frequent, and is commonly spoken of as cancer of the pleura, inasmuch as the microscopic picture it presents closely resembles cancer. It is usually a diffuse, wide-spread thickening of the pleura which resembles a chronic pleurisy or empyema during life. Secondary sarcomas and cancers often occur and do not require separate notice.

Treatment.—The treatment depends entirely on the results of the disease and the symptoms demanding relief. It is hardly necessary to remark that all measures used can only be palliative.

PARASITES OF THE PLEURA.

Hydatid disease is the only one of clinical importance. It usually invades the pleura secondarily to the liver, and presents the characters of a slow, insidiously-arising chronic pleurisy with effusion. The differentiation may be made by means of the exploring needle.

Treatment.—Hydatid cysts should be tapped, and if they refill tapped again. Usually simple tapping is all that is called for. Do not open the cyst freely unless its contents are really purulent.

Section 6.

DISEASES OF THE DIGESTIVE SYSTEM.

I.—*DISEASES OF THE MOUTH.*

INFLAMMATION OR STOMATITIS.

General Etiology.—It may be caused by some local irritant or by some general infection. The use of hot or unsuitable foods, the abuse of tobacco, and gastric disturbances are illustrations of the former; while the acute specific fevers are examples of the latter. Several varieties are recognised, the most important of which are—the simple catarrhal, aphthous, parasitic, ulcerative, gangrenous, suppurative, and chronic specific.

SIMPLE CATARRHAL STOMATITIS.

This is particularly common in children during dentition, but it may occur at any age.

Morbid Anatomy.—It may be local, involving part only of the tongue, lips or cheeks, or it may be general in the mouth. It is characterised by redness and swelling of the mucous membrane, with increased secretion. This exudate contains both leucocytes and desquamated epithelial cells, and is frequently rich in bacteria, mostly

ordinary atmospheric saprophytes. If it accumulates, it dries and forms a whitish or brownish coating or fur on the tongue or around the teeth. In severe cases small cysts or superficial erosions may be seen. In chronic cases local hyperplasias of the epithelial cells may give rise to silvery white spots, called *leukoplakia*, or to circinate patches of desquamated epithelium on the tongue, which spread at their margins, while they heal at their centres. They cause itching and heat. The disease is known as *eczema of the tongue*, or, when the tongue is covered with such areas, like a map, as *geographical tongue*.

Symptoms.—Local discomfort, amounting even to pain in the mouth, is all that is usually complained of.

The Treatment consists in removing the cause of the condition and correcting any gastric disturbance which may be present. When necessary, wiping or washing out the mouth with the glycerinum boracis tends to correct any fetor of breath.

APHTHOUS STOMATITIS.

This has the same characters as catarrhal stomatitis, but shows, in addition, a number of small opaque or white spots with red margins upon the lips, gums or tongue. They are often present in groups and may coalesce to form large areas. They frequently begin as vesicles, but sooner or later show a fibrinous exudate in and upon the degenerated epithelium. They are most common in young badly-fed and badly-housed children, but have been found also in women during menstruation, pregnancy and the puerperium. No special germ has been found in association with them.

Treatment.—Similar treatment to that given for simple catarrhal stomatitis should be carried out, but, in addition,

much relief will be obtained by touching any painful ulcer with nitrate of silver solution in distilled water (10 to 20 grains to the ounce) or with a modified lunar caustic pencil. It is essential to dry the surrounding mucosa before treatment with the solid caustic.

PARASITIC STOMATITIS OR THRUSH.

Parasitic stomatitis is distinguished from aphthous stomatitis in there being generally no increase in the buccal secretion and in the causation and characters of the spots. These are caused by the fungus called *oidium* or *saccharomyces albicans*, which forms a felted mass of branching filaments with terminal spores on the surface of and within the epithelial cells. It is only exceptionally that they invade the deeper structures. Its occurrence is favoured by the use of milk and starchy foods, hence it is common in young children and in adults debilitated by disease where sufficient attention is not paid to the cleansing of the mouth. It usually appears first on the tongue in the form of milky or pearly-white spots, which tend to increase in size and spread to the cheeks, and backwards to the pharynx, and may even extend down the œsophagus to the stomach. The patches may afterwards become brown or black if the mouth is not cleansed.

The Treatment consists in washing out the mouth with a solution of chlorate of potash, glycerinum boracis or other suitable antiseptic. In children, wiping the mouth with the pharmacopœial glycerinum boracis is frequently sufficient. Attention to the bowels is desirable, because in most cases of thrush the alimentary system is disordered.

ULCERATIVE STOMATITIS.

Ulcerative stomatitis is characterised by the presence of definite ulcers. There are many varieties, which differ

both in the amount of the general stomatitis and in the characters of the ulcers, according to the conditions under which they arise. The gums are more frequently attacked than any other part of the mouth. They are usually reddened and swollen, particularly around the teeth, which are apt to become loosened. In the variety known as *pyorrhæa alveolaris* (common in elderly people) there is little general stomatitis, and the inflammation seems to begin in the ligaments of the teeth and then to extend to the gums around the teeth, leading to swelling, retraction and absorption of the gums, which thus fall away from the teeth and cause the constant exudation of a small amount of pus around the teeth. In the variety known as *putrid sore mouth* or *fetid stomatitis*, which is common in young badly-fed and badly-housed children after the first dentition, and often spreads epidemically in institutions, there is, on the other hand, a marked general stomatitis, while the gums are swollen and red, projecting upwards between the teeth, which become loosened. The ulcers appear on the gums and spread along the gum line of the upper and lower jaws, rarely attacking the cheeks or tongue. They vary in size and depth, being mostly of only slight depth, but occasionally extending down even to the bone, which may become in turn superficially involved. This variety often behaves clinically as if it were a specific disease caused by a special micro-organism, but it is probably only a variety of ordinary ulcerative inflammation capable of being produced by various septic germs. The local and constitutional disturbance is often severe. Other varieties of ulceration may occur in mercurial and other forms of poisoning, in scurvy and other blood diseases, and in other cases which need not be specially mentioned.

Treatment.—The mildest form of ulcers should be treated in a manner exactly similar to that recommended for aphthous stomatitis.

The form described as *fetid stomatitis* in which the disease may spread and involve the bone, demands more vigorous antiseptic treatment. Wash out the mouth with strong chlorate of potash solution, or permanganate of potash solution, and the affected areas may be painted with powerful antiseptics, such as 1 in 3000 or 5000 corrosive sublimate solution. Attention must next be paid to the profound systemic disturbance. Iron should be administered, quinine is often of value, and alcohol should be freely given to support the patient's strength.

GANGRENOUS STOMATITIS—CANCER ORIS—NOMA.

This somewhat rare condition begins in the mucous membrane, most commonly near the angle of the mouth, as a red livid area which quickly ulcerates, the floor and margins of the ulcer showing reddish-black gangrenous tissue. In mild cases, which are exceptional, it soon ceases to spread, the sloughy tissue is cast off, and it heals spontaneously. In most cases it spreads, and presently reaches the skin of the cheek, which, at first red or black and gangrenous, soon becomes perforated, forming a gap in the cheek bounded by gangrenous walls of wider extent inside than outside. The tissues of the cheek for some considerable distance around are reddened and intensely œdematous. The gangrene spreads, the gap getting bigger, and may invade the nose, eyelids, ear, and jaws. Various germs have been found in the affected tissues, e.g., the diphtheritic or pseudo-diphtheritic bacillus has been found in some cases, Hoffmann's bacillus in some, a thread-like bacillus in others; but the bacteriology of the condition is imperfectly known. It is chiefly met with in debilitated children of tender years, mostly before six or seven years old, though occasionally up to twelve years old, who have been badly fed and housed. It may occur after any acute infection, but measles appears to be specially provocative,

as a large number of the recorded cases have come on during convalescence from this infective disease. General intoxication is naturally severe from the extensive toxin absorption. Pneumonia often supervenes. Death usually occurs within ten days.

Treatment.—Antiseptic treatment should be carried out in as rigorous a manner as in the preceding disease. Apply strong nitric acid to the surface of the ulcer, or nitrate of silver, and wash out the mouth with solutions of chlorate of potash, chlorine water, permanganate of potash, or other suitable antiseptic. Support the strength of the patient by tonics, especially iron and quinine, and give strong beef-tea and plenty of milk; in many cases it is necessary to order alcohol freely.

SUPPURATIVE STOMATITIS.

The commonest form is the alveolar abscess or gumboil, which arises from inflammation of the pulp of a tooth spreading to the periosteum and tissues of the gums surrounding the roots of the teeth. A much more serious form is that which follows suppurations or erysipelas of the face or traumatism of the lips or tongue, or in the course of Bright's disease.

Treatment.—The treatment of a gumboil is extremely simple. Attend to the bowels, use a simple antiseptic mouth-wash, and after a preliminary poultice to aid in the ripening of the gumboil early incision affords relief.

CHRONIC SPECIFIC STOMATITIS.

There are three varieties of this, viz., the tubercular, the syphilitic, and the actinomycotic. Tuberculosis forms reddish-yellow swellings, which break down, forming

irregular ulcers, mostly upon the tongue or lips. They are rarely primary, being most frequently secondary to tuberculosis of the lungs or lupus of the face. Syphilis occurs either as a primary chancre on the lips or, much more commonly, as secondary or tertiary lesions. The secondary lesions are simple and superficial and consist of small flat papules, the covering epithelium of which becomes of a pearly-bluish-white colour, or of superficial ulcers or fissures or radiating scars about the angles of the mouth. The tertiary lesion is the gumma, which occurs most frequently in the posterior wall of the pharynx, the palate, or the tongue, and is generally smaller than a bean. It leads to ulceration and cicatrization. Actinomycosis is far more frequent about the mouth, jaws or neck than in any other part of the body in man. The germ gets in by a carious tooth or through the mucous membrane of the mouth or pharynx. It may cause a swelling at the point of entrance, but more usually it is in the deeper tissues of the gums, jaws, cheeks or neck. The swelling is nodular and breaks down in time, discharging pus in which the typical colonies of the germ can be easily seen.

Treatment.—Scraping the ulcer in *tubercular* cases is the best form of treatment should the patient's health permit of such radical measures. In *syphilitic* cases, in addition to antisyphilitic treatment, the local application of mercury is often successful. In *actinomycosis* the treatment is purely surgical, although iodide of potash is believed to have a remarkable influence in certain cases.

1

II.—DISEASES OF THE SALIVARY GLANDS.

ALTERED CONDITIONS OF SALIVARY SECRETION.

It may be abnormally increased, a condition known as *ptyalism*, or diminished, known as *xerostomia*. The former occurs in certain mental, nervous and generative conditions, and also after certain drugs, notably mercury, the latter is more rare, and its causation is obscure.

Treatment.—*Ptyalism* may be relieved by the use of belladonna or its alkaloid atropin, and any causal factor, such as mercury, should be eliminated. In *xerostomia* no treatment other than palliative is possible, and as there is no saliva its place should be taken by the use of fluid along with solid food.

INFLAMMATION OF THE SALIVARY GLANDS.

There are several varieties, *e.g.*, (1) epidemic parotitis or mumps, *vide* page 181; (2) symptomatic parotitis, which occurs in fevers, notably typhoid, and in connection with disease or injury of any of the abdominal or pelvic organs; (3) angina Ludovici, a rare form of phlegmonous inflammation of the salivary glands and surrounding tissues of the floor of the mouth and neck. It is essentially a cervical cellulitis caused by infection from a carious tooth or some trauma in the floor of the mouth. Milder forms of inflammation of the ducts of the glands may lead to the formation of salivary calculi and cysts. The most typical cyst is the *ranula*, which is a retention cyst of the sublingual gland or of the small acinous glands situated on the under surface of the tongue near its tip.

Treatment.—In inflammation of the salivary glands, the carious tooth or other source of infection should be

treated. Where a *ranula* is present it may be incised, or by pressure its contents may be squeezed out.

TUMORS OF THE SALIVARY GLANDS.

Simple and malignant tumors occur. The most frequent of all is the mixed tumor composed of myxomatous, fibrous, and cartilaginous tissue. It is generally regarded as a sarcoma (an endothelioma).

Treatment.—Tumors, whether simple or malignant, should in most cases be removed.

III.—*DISEASES OF THE PHARYNX AND TONSILS.*

The tonsils are merely a part of the pharynx in which the lymphoid tissues are particularly abundant. They contain a considerable number of leucocytes, which may pass through the surface epithelium. These are strongly phagocytic, and in this way they may help to destroy germs, both on the surface and in the substance of the tonsils. The tonsils have been described as the sentinels of the alimentary system, and, like other sentinels, they may at times fail in their task and allow of the entrance of infection into a part or the whole of the body. Like the rest of the pharynx, they give rise to a considerable surface exudate when inflamed, which is apt to collect in the mouths of the follicles, where it produces a whitish or whitish-yellow colour, from admixture with desquamated epithelial cells. Inflammations which extend deeply into their substance cause such prominent tonsillar swelling and symptoms that it is customary clinically to distinguish tonsillitis as a separate affection, but this is unnecessary on pathological

grounds. Inflammations are the most important affections to which the general tissues of the throat are liable, and it is customary to speak of them as *angina* whether they affect the pharynx or tonsils. Both are usually affected, in all acute inflammations at any rate; but we speak of pharyngitis when the general pharynx is attacked, and of tonsillitis when the tonsils are mainly implicated. They are caused much in the same way as inflammations of the mouth, viz., by local irritation or a general infection, but particular emphasis may be placed upon the provocative influence of excessive smoking and overuse of the voice, and of such constitutional conditions as gout and rheumatism.

ACUTE PHARYNGITIS.

Morbid Anatomy.—The inflammation may be superficial or deep. The former is far the commoner. In it the mucous membrane is reddened and somewhat swollen, covered in whole or in part with a mucoid or mucopurulent exudate. In the latter—described as phlegmonous pharyngitis—the surface changes are not at first marked, but there is great swelling, which rapidly increases and comes to interfere with swallowing and respiration. Suppuration rapidly intervenes, constituting retropharyngeal abscess. Caries of the cervical vertebræ must also be remembered as a local cause of this abscess.

Symptoms.—In the former (catarrhal pharyngitis) there is a sense of discomfort in the throat, a feeling of dryness or tingling or pricking and even pain, particularly on swallowing. Hawking and cough may be complained of. There may be some deafness or hoarseness from implication of the mouth of the Eustachian tube or larynx respectively. The constitutional symptoms are slight, whereas in phlegmonous pharyngitis they (particularly the fever) are severe.

Treatment.—An ordinary sore throat is often cured without treatment at all, but the application of the glycerinum boracis, or in more severe cases of the glycerinum acidi carbolici diluted in two parts of glycerine, will afford much relief. Steam inhalations and the application of the wet pack externally to the neck are often efficacious. Attention should be paid to the Eustachian tube when deafness becomes marked and suggests the possibility of rupture of the tympanic membrane either from imperfect aeration or from middle ear suppuration.

CHRONIC PHARYNGITIS.

Morbid Anatomy.—Dilated venules are the most constant feature. The epithelium may be moist-looking and covered in places with a mucous exudate, or it may be dry and smooth (*pharyngitis sicca*). These patches may dry and form offensive scales or crusts. Small roundish, reddish projections may be studded singly or in clusters over the surface of the pharynx, giving it a granular appearance, hence the term *granular pharyngitis*. They are supposed to be due to hyperplasia of the lymphoid follicles.

Symptoms.—They are much the same as in the acute form, but less severe, dryness, pricking and hawking being the chief complaints. The granular form is sometimes blamed for interfering with the singing voice; but all forms of pharyngitis will do so to a certain extent.

Treatment.—Rest to the voice is often sufficient. Locally, apply astringents, such as the glycerinum acidi tannici or the glycerinum aluminis, and remember that change of air to a warmer and dry climate is most beneficial, particularly during the colder months of the year.

MEMBRANOUS PHARYNGITIS.

It may be caused by one of the ordinary inflammatory germs, such as the streptococcus, or the staphylococcus; but it is most often and most typically caused by the Klebs-Löffler bacillus, *vide* "Diphtheria" (page 96).

ULCERATION OF THE PHARYNX.

Ulcers occur in the pharynx in certain specific diseases, such as syphilis, tubercle and typhoid, and also accompanying ordinary chronic catarrh. In the last case they are small and superficial and called follicular. In syphilis they are also small and superficial during the secondary stage, but may be large and deep in the tertiary stage. They may lead to much cicatrization and deformity. Tubercular ulcers occur towards the later stages of pulmonary phthisis. Pallor of the mucous membrane is first seen, then one or more whitish-yellow foci which break down, forming ulcers with irregular, thickened margins. They generally cause much pain. In typhoid fever the ulcers vary in character. They may be caused by the bacillus typhosus or by one of the germs of ordinary inflammation.

Treatment.—The treatment must be on general lines, *syphilitic* and *tubercular* ulcers being attacked in the same way in which these affections are treated in the mouth.

ACUTE TONSILLITIS.

There are two varieties—the follicular and the suppurative.

FOLLICULAR TONSILLITIS.

Etiology.—The remarks made on the general etiology of inflammations of the mouth and throat apply to this

condition also, but experience has shown that young people exposed to bad hygiene, especially sewer gas, are particularly liable to contract the disease. It appears to be most common in spring and autumn.

Morbid Anatomy.—The tonsils are swollen and reddened, but it is mainly their surface layers which are affected. There may be some exudate over their surface, but it usually collects in their lacunæ, where it partly dries and gathers cocci and desquamated epithelial cells, forming whitish-yellow patches, at first small but later larger by confluence. It is often impossible to distinguish this form of membranous tonsillitis from diphtheria except by a bacteriological examination.

Symptoms.—Pain in the throat, particularly on swallowing, along with fever (often severe and rising rapidly to 105°F.) and general malaise are the chief symptoms. They generally subside within a week.

Treatment.—Remember that this form implies the presence of some infective organism, and therefore the application of the glycerinum acidi carbolici, or of a 1 in 3000 or 5000 solution of corrosive sublimate in water, will yield good results. The pain may be soothed by frequently washing out the mouth with warm boric lotion. Quinine is probably the best remedy internally. Feed the patient well, and advise rest in bed for a few days.

SUPPURATIVE TONSILLITIS OR QUINSY.

Etiology.—Similar to the last, but is most common in adolescents and young adults. One attack predisposes to another.

Morbid Anatomy.—The inflammation passes deeply into the tonsils and generally ends in suppuration. A

few cases resolve without pus formation, and a hard and fast line can hardly be drawn between superficial and deep inflammations, but cases mostly belong to either the one or the other. The term quinsy is used by some to designate any severe inflammation of the tonsils; but as ordinarily used it means a suppurative tonsillitis in which one tonsil generally, or if both, the one before the other, becomes enlarged and reddened. When both are affected at the same time, which is less common than only one, though as the one subsides the other may enlarge, they may meet in the middle line. In other cases the swollen tonsil presses the uvula over to the other side, and it as well as the mouth becomes coated with secretion. The glands and tissues below the jaw become enlarged, and the patient is not able to open his mouth except to a very limited extent, often hardly sufficient to allow of the insertion of the finger for the purpose of palpation of the tonsil. The tonsil becomes softer in a few days, usually three or four, when fluctuation can be felt. The abscess if not opened will burst spontaneously into the mouth or pharynx, giving the patient instant relief. It more rarely burrows deeply, producing gangrene, or eroding the internal carotid artery or one of its branches. If the pus enters the larynx it may produce suffocation. It rarely dries up without rupturing.

Symptoms.—Pain and dryness and swelling of the throat are first complained of. Marked fever and weakness follow, the temperature rising to about 105°F. Delirium is common, particularly at night. The local pain and discomfort become considerable, swallowing is painful and difficult, and liquids may not be able to be swallowed at all or may regurgitate through the nose. The patient has often a dread of impending suffocation, even where the swelling is far from filling the aperture of the fauces. It is only extremely rarely that it does so.

Treatment.—In cases where abscess formation follows an acute tonsillitis, early incision with a sharp-pointed knife, the greater part of the blade except the point being guarded with sticking-plaster, should be made, care being taken to avoid damaging the internal carotid artery. There is no fear of this happening if the incision be made in a vertical direction, the line being chosen midway between the pillar of the fauces and the extreme projection of the tonsil. The inhalation of steam, holding hot water in the mouth, and similar measures will aid the ripening of the tonsillar abscess.

Where it is possible to do so, try to abort the tonsillitis by giving quinine (10 grains in the day) or guaiac resin (1 to 3 grains thrice daily), and add a brisk saline purge in the morning. Remember the close association with rheumatism, and in such cases give the salicylate of soda in 20 grain doses. When necessary apply local antiseptics, such as the glycerinum boracis, to the surface of the tonsil: this treatment is specially desirable when there are follicular ulcers.

Iron and general tonics should be kept up for a considerable time, and every effort made by the use of astringents locally to reduce the size of the tonsils to their old dimensions. Many patients who suffer once from tonsillitis tend to have repeated attacks, and the condition may become chronic. In these cases it is wise to consider whether the tonsil or tonsils should not be excised, and care should be taken in children who are liable to the affection to avoid all muffling of the throat, and to sponge the neck and chest daily with cold water as a prophylactic measure.

CHRONIC TONSILLITIS.

The lymphoid tissues of the tonsil and of the vault of the pharynx become enlarged, sometimes greatly, producing *hypertrophy of the tonsils* and adenoids.

Etiology.—It is most common in adolescents and young adults. It is probably the result of preceding catarrh.

Morbid Anatomy.—The size of the enlarged tonsils varies greatly. They often meet in the middle line. The enlargement may involve chiefly either the lymphoid tissue or the stroma, or both. It is generally accompanied by considerable adenoid growths, though these latter may be large without much enlargement of the tonsils themselves. They generally form soft papillomatous masses more or less filling up the posterior nares and the vault of the pharynx. They may press upon or cause inflammation in the Eustachian tubes, resulting in deafness.

Symptoms.—The patients breathe through the mouth and may show a peculiar vacant expression of face. They may complain of headache and earache. The sleep is disturbed by the snoring and snorting breathing. Speech becomes nasal. Apathy and mental dulness become marked, and in long-standing cases a peculiar deformity of the chest appears. The pigeon or chicken breast is the commonest form, but a barrel or funnel shape is sometimes seen.

Treatment.—In many of these cases there are adenoids in the naso-pharynx, and the best treatment is to remove both tonsils and adenoids as soon as possible. Where this is unnecessary, the application of powerful astringents may yield good results, and remember that Mandl's solution, which consists of varying strengths of iodum ($6\frac{1}{4}$ to 20 grains), potassium iodide (25 to 75 grains) in 1 oz. of glycerine, in addition to a few minims of the oil of peppermint, if applied two or three times a day for a period of three to four weeks to the tonsils, often yields excellent results.

IV.—*DISEASES OF THE ŒSOPHAGUS.*SUB-GROUP (a)—*ALTERATIONS OF THE LUMEN.*

The two most important alterations are pouching and narrowing of the lumen.

DIVERTICULUM.

Pouching or diverticulum is of two varieties, known as the *pressure* form and the *traction* form respectively. The former occurs at the junction of the pharynx and the œsophagus, and is due to pressure from within upon a weakened muscular wall. The food collects there and increases the pressure, so that in time a saccular bulging is formed by the mucosa being forced outwards through the weakened and separated muscular fibres of the muscular coat. It may reach a large size. The latter—the traction form—is much smaller and forms one or more conical sacs at the level of the bifurcation of the trachea. The apex of the sac is often adherent to contracted and diseased lymphatic glands. The latter during their earlier enlargement become adherent to the outer wall of the œsophagus, and during their subsequent contraction pull it after them.

Symptoms.—Dysphagia and regurgitation are the chief complaints. The diverticulum can often be determined by means of the œsophageal bougie. At one time the bougie passes freely into the stomach, at another it stops abruptly through its having entered the diverticulum.

The Treatment is surgical. In many cases the diverticulum can be removed, although where it is the result of malignant stricture no operative interference is possible, and recourse to the œsophageal tube may be necessary for the feeding of the patient.

STENOSIS.

Narrowing of the lumen or stenosis or stricture is the result of pathological processes arising within the lumen, within the walls or outside them. Foreign bodies, such as coins, fish bones, &c., may stick in the tube and cause inflammation or ulceration, which leads to cicatrisation, and hence stricture. Cancer arises within the walls of the tube, and causes obstruction first by the tumor mass to which it gives rise and later by the cicatrisation which follows the ulceration of the tumor. Aneurism and tumors of the mediastinum cause stricture of the œsophagus by pressure upon it from without.

Symptoms.—Inability to swallow, particularly solid food, and regurgitation of food are often marked. The œsophageal bougie must be used with care, and not at all in cases where an aneurism is suspected. It is of great use, however, in the spasmodic stricture, often called œsophagismus, which is met with in neurotic individuals. The bougie may meet with no resistance, or with a slight resistance which gives way gradually, showing that no real or organic stricture exists.

Treatment.—In cases which are not malignant, an attempt may be made to dilate the stricture by the passage of a bougie. Sometimes in malignant cases the passage of a soft œsophageal tube or the introduction of a vulcanite tube temporarily into the stricture may produce much benefit by mechanical dilatation and by relieving spasm. In most malignant cases it is necessary sooner or later to perform gastrostomy. Antispasmodics, such as dilute hydrocyanic acid (3 minims), will be found useful, while recourse may be necessary to opium or morphia where pain is excessive.

SUB-GROUP (β)—INFLAMMATIONS OF THE
ŒSOPHAGUS.

ACUTE ŒSOPHAGITIS.

Etiology.—This comparatively rare condition is met with after swallowing irritating or corrosive fluids or solids, or in certain acute infections, *e.g.*, typhoid, small-pox, pyæmia, &c.

Morbid Anatomy.—The mucous membrane is swollen and the epithelium desquamated. Actual denudations or ulcers of various sizes may be present. In the severe variety—known as *phlegmonous*—there is purulent infiltration of the submucosa, giving it a white appearance.

Symptoms.—Pain on swallowing, spasm and regurgitation of food may be present, or there may be no symptoms.

Treatment.—Sedatives should be given, and bland, soothing drinks, such as barley water, are frequently comforting to the patient. In the case of severe inflammation, rectal feeding gives a certain amount of rest to the inflamed part. The later treatment depends on the development of stenosis.

CHRONIC ŒSOPHAGITIS.

It is doubtful if there be such a thing as chronic œsophagitis; but some authorities hold that it is sometimes present, particularly in catarrhs of the cardiac end of the stomach, and that the dilated veins found at the termination of the œsophagus, which occasionally rupture, particularly in cases of cirrhosis of the liver, are a manifestation of this condition.

SUB-GROUP (γ)—CANCER OF THE ŒSOPHAGUS.

Certain benign tumors, *e.g.*, papilloma, lipoma, and myoma, occasionally occur; but the only tumor of real practical importance is cancer. It is almost always a squamous epithelioma originating in the covering epithelium, though a glandular cancer originating in the mucous glands may occur. It may occur at any part of the tube, some statistics putting it commonest in the lower third, and others in the upper third. It is common also at the level of the bifurcation of the trachea. It forms at first a swelling of the mucous membrane which projects as a tumor into the lumen. After a time it ulcerates and then has the appearance of an ulcer with thickened infiltrated margins spreading like a ring around the œsophagus. This causes obstruction, hence the tube dilates and its walls hypertrophy above the seat of the cancer. The degree of stricture becomes steadily greater, and the ulcer may eat through the wall of the tube into the trachea, a bronchus, the lung, or any of the neighbouring tissues.

Symptoms.—Difficulty in swallowing is first experienced. It tends to steadily increase until ulceration occurs in the tumor, when it may be considerably relieved for a time, but it sooner or later returns and steadily progresses till death. Solids and liquids may both be able to be swallowed for a time, but ultimately only liquids can be got down. Regurgitation of the food is another symptom. It comes on later in the disease, as a rule, and may occur immediately after the food is taken, particularly when the cancer is high up, or be delayed for some minutes. Pain may be present at other times than during swallowing and may be very great. Emaciation is one of the most marked of the later symptoms. It becomes, indeed, more

pronounced in this than in any other disease. The glands of the neck may be enlarged even early in the disease. Death is certain, and is due either to asthenia or to some complication brought about by the perforation of the ulcer, or in some other way.

Treatment.—Much which has been stated under the head of stenosis might be repeated here. The passage of an œsophageal tube or the temporary introduction of a vulcanite tube frequently proves of much benefit. Pain may be combated by the use of opium and other sedatives where necessary.

V.—DISEASES OF THE STOMACH.

DYSPEPSIA.

DEFINITION—Any disturbance, such as pain or discomfort, occurring during the process of digestion is generally spoken of as dyspepsia or indigestion.

Etiology.—It is generally discussed under diseases of the stomach. This is convenient but a little misleading, inasmuch as many cases of dyspepsia are due to functional disturbances in the process of digestion, no organic disease being present either in the stomach or elsewhere. Further, the process of digestion is a complicated one, performed partly in the mouth, partly in the stomach, and partly in the intestines, and many of its disturbances are referable either to the mouth, the pancreas, the liver, or the intestines, and not to the stomach. Food consists of proteids, carbohydrates and fats. Oral digestion is chiefly mechanical, the food being triturated, disintegrated and mixed with saliva. The *ptyalin* ferment contained in the saliva acts

upon the carbohydrates of the food in the mouth, and after its introduction into the stomach, until the hydrochloric acid reaches .003 per cent., when it ceases. The main action of the gastric juice is upon proteids. It contains the ferment *pepsin*, which acts only in the presence of free hydrochloric acid. It first converts the proteids into acid albumins, next into primary albumoses, then secondary albumoses, and finally into diffusible amphopeptones. It has no action on fats. The partially digested food begins to leave the stomach after about two hours, and has generally all done so after about five hours. Its entrance into the duodenum stimulates the flow of pancreatic, biliary and intestinal fluids, and as soon as its acidity has been overcome by the alkalinity of these fluids, intestinal digestion begins. The pancreatic fluid plays the most important part in this digestion, indeed in the whole digestive process. It acts upon all the food constituents—the proteids, the carbohydrates and the fats. The proteids are converted by means of its *tryptic* ferment, which acts only when the medium is alkaline, into secondary albumoses, then into amphopeptones, and then partly into antipeptones, which are diffusible and absorbed, and partly into hemipeptones, which undergo further conversion into certain end products, such as leucin and tyrosin. The carbohydrates are acted on by its *amyllopsin*, a ferment apparently identical with the similarly acting ferment of the saliva (ptyalin), and converted through several intermediary steps into maltose. The fats are digested by another pancreatic ferment named *steapsin*, aided by the bile. They are probably split into glycerine and fatty acids, the latter combining with the alkaline salts of the bile and intestine to form soaps. Both glycerine and soaps are readily diffusible and absorbable. The bile and the intestinal juice proper—the *succus entericus*—help the pancreatic juice. The former helps it to neutralise the acid chyme coming from the stomach, and to emulsify fats, the latter helps it to digest carbo-

hydrates, for it contains not only an amylolytic ferment similar to that of the pancreas, but also a special inverting ferment called *invertin*, which transforms the maltose into dextrose. This very short résumé of the essential facts in digestion is useful, as it shows how complex the process of digestion is, and how it may be disturbed through some faulty step in the mouth, stomach or intestine, the last including also both the pancreas and the liver. Such faulty step may arise through mere disordered function without any organic disease, or it may be caused by organic disease; but whether it is due to the one or the other the term *dyspepsia* is used to designate such mischief when certain prominent symptoms of disordered digestion, presently to be mentioned, form the chief trouble of which the patient complains. In the mouth, for instance, the chief fault is imperfect mastication of the food, either from too rapid eating or imperfect teeth. It has been said that each mouthful of solid food requires at least twenty-five bites before it is triturated sufficiently to be swallowed, but it is safer to say that no food should be swallowed until it has been completely disintegrated by chewing and until it has been thoroughly mixed with the saliva into a soft pulp by means of the teeth, tongue and buccal muscles.

Disturbances of gastric digestion may arise from faulty secretion of pepsin or of hydrochloric acid. The former only occurs in those rare cases in which the gastric mucosa is completely atrophied, or exceptionally in certain nervous conditions, and it may be disregarded as a cause of *dyspepsia*. The latter, on the other hand, either in the form of deficiency or excess, is of frequent occurrence and requires consideration. It has been argued that inasmuch as the stomach is intended mainly for the digestion of proteids, in which it plays only a subsidiary part (the pancreas being able to do this completely even without its help), disturbances of gastric digestion need not cause any trouble. Actual clinical experience does not bear

this out, for disagreeable symptoms are often found to follow upon disturbances which appear at any rate to be of purely gastric origin. It is easy to understand why this should be so. If the hydrochloric acid be deficient, as it generally is in inflammations, degenerations and cancers of the stomach and profound anæmias, and occasionally is in *tabes dorsalis* and neurasthenia, the food may be retained within the stomach for an abnormally long time. Abnormal fermentations, both gaseous and acid (lactic, butyric and acetic), may then arise, for the various bacteria which enter with the ingested food, instead of being destroyed, live and multiply. Irritative and toxic substances are thus produced which may cause vomiting, gastric distress, sleeplessness, and other disagreeable symptoms. When gastric digestion is normal, the bacteria introduced with the food succumb in the gradually increasing acid medium, so that the healthy fasting stomach is always sterile. If, in addition to the diminution in the hydrochloric acid, there be also some obstruction at the pylorus or some weakness in the stomach muscles, these symptoms become more pronounced. If the hydrochloric acid be excessive, which it may continuously be as a chronic condition in gastric ulcer and in oft-repeated dietetic errors, or as a more acute intermittent condition in rare cases of *tabes dorsalis* and profound neurasthenia, the mucous membrane is thereby irritated, and acid eructations, vomiting, gastric distress and other symptoms may follow. The intermittent variety may come on independently of food, and an attack may last several days, vomiting of a clear, watery, burning fluid being its most striking and persistent symptom. The chronic variety, on the other hand, is more common, and its symptoms usually come on after food, but occasionally in the fasting stomach, as shown by the appearance of acrid eructations or vomit during the night or early morning. Disturbances of intestinal digestion may arise from faulty gastric action (the food

leaving the stomach at unusual intervals or in an imperfectly digested state) or from faulty pancreatic, biliary or intestinal secretions. It has already been stated that this (the intestinal) is the most important part of the digestive process. All the foods, viz., proteids, carbohydrates and fats, are here digested, the first two mainly and the last entirely, and disturbances therein are likely to be frequent and to have disagreeable consequences. Many dyspepsias, perhaps the majority, are due to faulty intestinal digestion, but owing to the complexity of the process, the pancreas, liver, intestine, and even stomach, all being involved, it is often difficult or impossible in the present state of our knowledge to trace exactly what the fault is and what it is due to. Further, it must be noted that dietetic errors, both as to the kind and quantity of the food and drink, and irregularities in meal times, nervous influences, *e.g.*, worry, chills, want of exercise and weakening disease, may have an important bearing upon the causation of dyspepsia.

Symptoms.—The symptoms characteristic of dyspepsia are a furred tongue, a bad taste in the mouth, disagreeable breath, a failure of appetite, nausea, vomiting, a sense of oppression in the chest or weight in the stomach region, actual pain (varying greatly in extent and character), pyrosis or waterbrash (eructation of a quantity of a colourless, slightly alkaline, opalescent fluid), acidity due to hydrochloric, lactic, butyric or other organic acid, flatulence (causing distension), belchings or borborygmi, constipation or diarrhœa.

Treatment.—The treatment falls under different heads, depending on whether the fault lies with the mouth, stomach or intestines, or with the food or the patient's general health. The teeth should be carefully investigated and the habits as regards proper mastication, time allowed

for meals, and for digestive processes thereafter, demand supervision. The dietary is important, and especially the quality, quantity and the satisfactory preparation of the food as regards its digestibility. Affections of the stomach and intestines are considered elsewhere, but the condition of the mouth may also call for treatment, and especially where there is inflammation of the gums in connection with carious or imperfectly brushed teeth or where some form of stomatitis is present. The patient's general health, if below par, may require the administration of tonics, such as the mineral acids and strychnin.

SYNOPSIS of the Causes and Symptoms of Dyspepsia.

CAUSES A.—

1. Imperfect mastication.
2. Mental exertion, active exercise—after a full meal.
3. Irregular arrangement of meals.
4. Insufficiency of food.
5. Injudicious mixture of foods.
6. Indigestible or unwholesome foods.

CAUSES B.—

1. Catarrhal inflammation and congestion of mucous membrane of stomach.
2. Gastric ulcer.
3. Cancerous and other morbid growths in stomach.
4. Dilatation of stomach.
5. Diminution of stomach.
6. Degenerative changes of mucous membrane of stomach, due to—
 - (1) Alcohol.
 - (2) Catarrh.
 - (3) Derangement of gastric secretions.

CAUSES C.—

1. Constipation.
2. Impairment of function from pressure of other organs.
3. Impairment of function due to lesions of other organs.
 - (1) Liver and portal system.
 - (2) Heart.
 - (3) Lungs.
 - (4) Kidneys.} Causing congestion.
4. Impairment of function due to disease of the nervous system.
 - (1) Mental emotions, grief, &c.
 - (2) Reflex—*i.e.*, pregnancy.
5. General Diseases—Gout, phthisis, fevers, anæmia, &c.

SYNOPSIS—*Continued.*

SYMPTOMS.

I. REFERABLE TO STOMACH—

- (1) Appetite.
 - (a) Capricious.
 - (b) Loss.
 - (c) Ravenous.
 - (d) Depraved.
- (2) Abdominal sensations.
 - (a) Simple uneasiness.
Feeling of weight, sinking, fulness.
 - (b) Pain more or less acute.
- (3) Flatulence.
- (4) Eructation.
- (5) Nausea and sickness, and even vomiting.
- (6) Pyrosis.

2. REFERABLE TO OTHER ORGANS—

- (1) Tongue.
 - (a) Clean.
 - (b) Furred, flabby, pale, swollen, &c.
 - (2) Bowels.
 - (a) Constipation.
 - (b) Diarrhœa.
 - (3) Heart—Palpitation, irregular, quickened, intermittent.
 - (4) Nervous system—Headache, vertigo, hypochondriasis, neuralgia, depression of spirits, &c.
 - (5) Skin—Eczema, urticaria, erythema, &c.
 - (6) General system—Emaciation, &c.
-

DEGENERATIONS OF THE STOMACH.

CLOUDY SWELLING—FATTY DEGENERATION—
INFILTRATION.

Cloudy swelling, fatty degeneration and infiltration occur in the glands of the mucosa under the same conditions as in other glandular organs, particularly in the severe infections.

WAXY DEGENERATION.

Waxy degeneration occurs in the mucosa, and more markedly in the muscular coats, affecting the walls of the vessels and connective tissue fibrils here as elsewhere.

CALCIFICATION.

Calcification of the stomach in the form of white, gritty patches upon its inner surface is of rare occurrence. It has been met with in cases of poisoning with mercuric chloride, in which the lime salts have been absorbed from the bones and deposited in the stomach.

It is important to remember that these degenerations do occur and that they will influence gastric digestion; but they do not require a more detailed description, as they always form a comparatively insignificant part of a more general disease.

MALFORMATIONS AND DISPLACEMENTS.

Only the more important need be noticed.

STENOSIS.

Total or partial (more frequent) stenosis of the pyloric orifice may be congenital or acquired. It leads to dilatation and hypertrophy of the stomach.

HOUR-GLASS DEFORMITY.

Hour-glass deformity of the stomach usually shows the constriction about the middle. It may be congenital or acquired and due to persistence of a physiological contraction, the cicatrization of a simple or malignant ulcer, or to adhesion to displaced viscera.

DISPLACEMENT OF THE STOMACH.

In the embryonic condition the stomach is placed nearly vertically, and this position is often found in the adult. Some authorities believe that this is always the normal position, the cardiac part (the true stomach) being vertical and the pyloric (nearly cylindrical in shape) being horizontal or curving somewhat upwards. The whole stomach may be displaced either upwards, forwards (in deficiencies of the diaphragm or abdominal wall) or downwards. The last is the most common and important. It may be caused by pressure from above, as from corsets, an enlarged liver, &c., or by traction from below, as from an overloaded colon or the traction of adhesions. It varies in degree, and is believed to be due to relaxation of the gastro-hepatic and hepato-pyloric ligaments. It therefore results in a displacement downwards of the pyloric portion of the stomach, a condition known as *gastroptosis*. It is usually associated with a displacement downwards of the transverse colon (*enteroptosis*), right kidney and liver. Individuals in whom this occurs are very liable to a certain form of nervous dyspepsia, or even neurasthenia, but the relationship is not clearly understood.

INFLAMMATION OF THE STOMACH— GASTRITIS.

Varieties.—The inflammation is usually simple and superficial, involving the mucous coat only, when it is termed *gastric catarrh*; at other times it extends more deeply into the coats, when it is termed *toxic* if caused by poisons, *infective* or *mycotic* if due to bacteria. A particular variety of the last is *phlegmonous gastritis*, in which there is a suppurative inflammation involving specially the submucous coat; another is *membranous* or *pseudo-membranous gastritis*, in which a fibrinous or muco-fibrinous exudate collects upon the surface of the stomach as a more or less perfect covering.

SIMPLE ACUTE GASTRIC CATARRH.

Causes.—The most common cause is some error in diet. Indigestible food, or even an excess of food, and over-indulgence in alcohol are among the commonest of such errors. It is favoured and may even be caused by poisons circulating in the blood, hence it occurs in the ordinary infective fevers, either during the attack or during convalescence. It is more common in children, in whom it is often accompanied by some intestinal catarrh, than in adults.

Morbid Anatomy.—Redness and swelling of the mucous membrane of more or less general distribution, with an increase of mucus, are the chief changes. They vary greatly in degree, according to the severity of the irritant, and are apt to largely disappear after death. The microscope may detect morbid changes even when the macroscopic changes have disappeared. The chief microscopic characters are dilatation of the blood-vessels of the mucous coat, infiltration of the interstitial tissue with fluid and leucocytes, cloudy

swelling and desquamation of the cells lining the gastric glands, and great increase in the number of goblet cells. The mucus covering the surface of the stomach contains leucocytes and desquamated gland cells. The mouths of the gastric glands or the surface of the solitary follicles are sometimes eroded, forming small superficial ulcers, and occasionally in severe cases there are small hæmorrhages.

Symptoms.—Two clinical types are recognised—a moderate non-febrile type and a more severe febrile type: The former begins with discomfort or pain in the gastric region, headache, nausea, eructations and vomiting, which usually affords relief. The nausea and vomiting may soon return and be repeated at short intervals, particularly in children, in whom there is often also diarrhœa and colic. In rare instances in children the attack is ushered in by aphasia, asthma or epileptiform convulsions. The duration of this mild form seldom exceeds twenty-four to thirty-six hours. The febrile type, sometimes spoken of as “gastric fever,” usually begins with a chill, shivering, headache and pains in the limbs. The temperature rises to 102° or 103°F . The tongue is furred, appetite is lost, and thirst may be very great. Vomiting is frequent but inconstant, and is apt to recur again and again, the later vomited matter consisting mainly of bile and mucus with no free hydrochloric acid. Constipation is more common than diarrhœa. The pulse is quick and weak and the urine scanty and high coloured. Sleep is disturbed. The attack usually attains its maximum by the third day, and may terminate abruptly, but usually it takes several days more to disappear.

Diagnosis.—It is usually easy, but may be difficult in the febrile cases which appear to arise spontaneously and cannot be referred to any error of diet. It may be impossible for some days to distinguish between it and

typhoid or other infectious fever, but a careful examination of the fever and other symptoms will generally enable this to be made in time.

Treatment.—In the milder type the evacuation of the irritant is often followed by rapid subsidence of the gastric catarrh. It is prudent, however, to give milk diet or beef-tea for one or two days, and to prescribe bismuth subnitrate (10 to 30 grains) with dilute hydrocyanic acid (3 minims), and when the ingestion of simple food is followed by local discomfort to prescribe pepsin with dilute hydrochloric acid, or to give the food pre-digested. It is often judicious to afford a period of rest to the organ, and in any case to feed at considerable intervals of time. In the febrile and more severe cases, rectal alimentation is called for, with complete rest to the stomach. Ice to suck allays hunger and gastric pain, and only after the attack has subsided should milk or beef-tea, generally pre-digested, be cautiously administered by the mouth. Thirst should be allayed by sipping lemon juice in iced water. Later, alkaline mineral waters with milk are often ordered. Where there is much pain, a linseed poultice or mustard leaf, applied over the region of the stomach, affords great relief. If vomiting or retching is excessive, lavage is one of the best methods of checking it, and as a rule the stomach should be first washed out with a warm alkaline solution, such as sodium bicarbonate (1 or 2 ounces to the pint), to dissolve the glairy mucus, and thereafter with weak permanganate of potash. Morphia is sometimes indicated, and $\frac{1}{8}$ to $\frac{1}{4}$ grain may be administered hypodermically, or as a suppository ($\frac{1}{2}$ to 1 grain) by the rectum.

SIMPLE SUBACUTE GASTRIC CATARRH.

Causes.—It is more common than acute catarrh and is caused in the same way.

Symptoms.—Frontal headache and epigastric pain are the earliest symptoms. Flatulence, nausea, and vomiting usually supervene. The vomiting only occurs at intervals or after an unusually full meal, and the vomited material shows an excess of mucus and a diminished percentage of hydrochloric acid. The appetite is impaired, and though at first the patient may be able to take an ordinary mixed diet, he soon begins to restrict its scope, usually first rejecting fats and sweets, then other articles, until only milk and broth are partaken of, and yet the symptoms remain. The stomach is usually dilated, the complexion pale or sallow, and the individual listless and apathetic. The bowels are usually constipated and the stools clay-coloured. The duration of the disease is uncertain. In young people it may come to an end comparatively soon if appropriately treated, but in the middle-aged it may last a long time.

Treatment.—Rest to the stomach is sometimes necessary, but pre-digested food given at intervals of four to six hours, and in moderate quantities, may be sufficient. Any diminution in the normal amount of hydrochloric acid should be made up artificially, and that alone may prove successful. Dilatation of the stomach is certainly best treated by lavage, and then by giving milk in steadily increasing quantities until several pints are assimilated without inconvenience to the patient. Clay-coloured stools indicate sluggish action of the liver, and the addition of phosphate of soda to the milk is beneficial, with an occasional dose of calomel or other more vigorous cholagogue purge.

SIMPLE CHRONIC GASTRIC CATARRH.

Etiology.—It may follow after repeated acute or sub-acute attacks, or may be chronic from the beginning, and from the same causes. Dietetic errors are particularly common and effective causes. They are too many for

mention, but indulgence in indigestible foods and in alcohol are among the most frequent. Excessive use of tobacco is another common cause of chronic gastric catarrh. Local conditions of the stomach, such as chronic congestion from cirrhosis of the liver or chronic heart disease, ulcer or cancer of the stomach, strongly favour, if they do not actually excite, catarrh; while certain constitutional states, such as anæmia, tubercle, Bright's disease and gout, act in the same way.

Morbid Anatomy.—The macroscopic changes may be so slight as almost to escape notice. The most constant are a quantity of tenacious mucus lining the surface and a thinning and irregular congestion of the mucous coat itself. There may also be little erosions thereof, with slate-coloured or reddish spots indicative of old or more recent hæmorrhages. The microscope shows a combination of parenchymatous and interstitial change, though either may greatly predominate. The former consists in granular and mucoid degeneration and atrophy of the surface and the gland cells, and the latter in new formation of fibrous tissue between the glands. In most cases of chronic simple catarrh the glandular degeneration is the chief change, the interstitial change being partly compensatory and partly inflammatory. The mucous coat in time thus becomes thinner and firmer. Some of the tubes may become dilated into small cysts owing to their outlet being obstructed. The interstitial change is either cellular or fibrous, according to its age. It is unequally distributed, being usually greater towards the pyloric region, and may thus pinch up the mucous membrane more or less extensively in places into swollen patches and even polypoid projections, causing it to assume a wrinkled, mammillated and polypoid appearance, a condition which has been described under the names of *état mammeloné* and *gastritis polyposa*. The common form of simple chronic gastritis, if it lasts long enough, results in producing a stomach with a somewhat

dilated cavity and a thinner, firmer wall, in which the microscope discloses a greatly atrophied mucous coat showing few remains of gland cells in a fibrous matrix and a fibrously infiltrated submucous and muscular coat. In rare cases, while the mucous coat may be atrophied to a certain extent, the interstitial change in it, but more particularly in the deeper coats, may be so excessive as to produce a great thickening of the whole wall of the stomach, which feels firm and leathery, while its cavity is usually much reduced. In typical cases of this rare disease the stomach has been likened to a *leather bottle*. Some of these cases follow upon a diffuse infiltration of cancer in the wall of the stomach, but others apparently follow upon simple catarrh, being a true and simple sclerosis.

Symptoms.—The leading symptoms are much the same as those of chronic dyspepsia, and in many cases it is impossible to say whether we are dealing with a stomach merely irritated by some functional disorder of digestion or one which is really inflamed. The symptoms vary greatly and are likely to be very persistent. They mostly come on after food. The tongue is furred, though the fur is often restricted to the dorsum, the tip and margins being red; or the whole tongue is swollen and red, its margins being indented by the teeth. There is often a bad taste in the mouth, and the smell of the breath may be disagreeable. The appetite is variable. It may be good, particularly early in the disease, but it is usually diminished, and in long-standing cases may be almost absent. In some cases it may be depraved, there being an abnormal craving for unsuitable food. Thirst is not often present, except in cases in which the catarrh has resulted in dilatation of the stomach. The most important symptoms are more or less directly related to the stomach itself. *Discomfort* comes on either immediately after the ingestion of food or, in the milder cases, not for an hour or two. It may be merely an

uneasy feeling, a *sense of weight, oppression* or *distension*, or it may be actual *pain*, more or less severe, of a burning character, localised to the region of the stomach, or more diffuse and often going through to the back. It may be felt chiefly in the chest—heartburn. It may gradually disappear without vomiting, or get worse and worse until relieved by spontaneous or voluntarily induced vomiting. *Epigastric tenderness* is more variable than the pain, but can generally be elicited at intervals in the course of the case. Deep palpation brings it out best, and it is usually felt all over the stomach, though there may be one or more painful spots. *Nausea* is an early and frequent, though by no means constant, symptom. It may be present only in the morning. It usually precedes vomiting, but may exist without vomiting, and comes on either immediately after food or more commonly one or two hours after. *Flatulence* may be, but is not usually, the chief symptom complained of. It varies greatly in amount, and is most marked in dilated stomachs. There may be also *eructations of gas* and small quantities of *fluid* (waterbrash). *Vomiting* comes on sooner or later in the course of the disease. At first it is only of occasional occurrence about two hours after a meal, and often ascribable to some particular error of diet. It may occur particularly in the morning, both in alcoholic and non-alcoholic subjects. It tends to occur with greater and greater frequency until, in neglected cases, it may come on more or less immediately after every meal, or be voluntarily induced by the patient to relieve the pain. There may be a good deal of retching before the vomiting, but there is rarely any after it. It is of the greatest importance to test the vomited matter in all cases as to the amount of *free hydrochloric acid* it contains and as to the presence of *mucus* and the *products of digestion*. In early cases of chronic gastric catarrh the percentage amount of free hydrochloric acid may be increased, but presently it becomes diminished, and in advanced cases with much

atrophy of the mucous membrane it may be entirely absent. Its diminution is often accompanied by the presence of free organic acids, e.g., butyric and acetic. The amount of mucus varies greatly. In some cases it is absent, in others it is present only in the form of a few pellets, and in others it is so abundant that the vomited matter is very tenacious and cannot readily be poured from one vessel into another. It is always accompanied by a diminution in the amount of free hydrochloric acid. When abundant it coats the surface of the stomach and prevents the flow of gastric juice and its free admixture with the food, so that treatment must be directed particularly to it. The presence of albumoses and peptones indicates a certain degree of activity of the gastric juice, and their amount relative to the time is a fair indication of the extent of disturbance of gastric digestion. *Hæmatemesis*.—Blood in small quantity is not infrequently present in the vomited matter. It may be only in streaks, particularly after much retching, or amount to an ounce or two. It is usually red, not like coffee grounds. *Constipation* is much more common than *diarrhœa*. The *urine* is scanty, high-coloured, and deposits many phosphates. There is rarely any albumen, though there may be a diminished excretion of chlorides. The *general nutrition* suffers. The face becomes pale and sallow looking. Anæmia may become marked. Headache and vertigo are common. The spirits become low and depressed.

Diagnosis.—The greatest difficulty lies in distinguishing chronic catarrh from merely functional dyspepsia. The attempt should always be made, though it may not always be successful. The presence of mucus in any quantity points to catarrh, so also does a continued diminution in the amount of free hydrochloric acid. These can be determined after a test meal. Gastric pain and vomiting after food are also strongly suggestive of

catarrh. In the middle-aged and old, when chronic catarrh remains persistent and does not yield to treatment, cancer should always be carefully looked for.

Prognosis.—It is essentially a chronic affection, though it is liable both to periods of improvement and of increased severity. In the young the outlook is good, but in the middle-aged and old, and in cases complicated with other disease, such as tuberculosis or Bright's disease, it is not so good. Much, however, depends upon a steady perseverance with suitable treatment.

Treatment.—Before attempting treatment we must try to make a diagnosis of (1) the condition of the stomach, whether there is any serious organic change present or not, and of (2) the cause to which the catarrh is due. Such diagnosis is by no means always easy, and a careful examination of the stomach contents should be a preliminary step in most cases.

The following points must also be investigated:—the quality and quantity of fluids and solids ingested, the time allowed for meals, the sufficient or insufficient mastication of food, and in particular whether the patient is partaking of any article of diet indigestible in itself or indigestible owing to some personal idiosyncrasy.

Where there are organic changes in the stomach, lavage and prolonged and careful dieting may be requisite to ensure comfort. To give a rigid milk diet is an admirable method of beginning treatment, and phosphate of soda or an alkaline mineral water is often added to the milk. Plain soups should replace milk where the latter is badly borne, and in any case the milk or soup may require to be peptonised in the first instance. There are certain articles of diet which should be avoided or taken in great moderation. These include butter and fat, potatoes, sugar and sweet things generally, new bread, and fruits with many seeds.

Once improvement has commenced, a gradual increase in amount and variety of food is not merely warranted, but helpful. If the gastric juice is deficient in free hydrochloric acid, order 10 minims of the dilute acid, with or immediately after food ; while if it is in excess give the dose about half an hour before food, so as to diminish the natural but excessive flow. On the other hand, an alkali given before food increases the natural secretion of hydrochloric acid, and given after food acts by neutralising any excess. Where organic acids are present in great amount there is generally deficient hydrochloric acid, and, in addition, sarcinæ or other fungi are in evidence. In such cases lavage is called for. Pepsin is less frequently deficient than is the hydrochloric acid, but it must sometimes be added. It is desirable to watch the evacuations so as to see if any undigested food is being passed, and if so to lessen the quantity taken or increase the amount of the digestive agent. Curdled milk is very readily detected in the stools.

Many remedies have been tried in cases where the stomach wall shows advanced pathological changes, but none are of any great advantage save to allay vomiting, discomfort and pain. Dilute hydrocyanic acid, bismuth and the other members of the sedative group are certainly invaluable, but silver nitrate, permanganate of potash, &c., are of doubtful benefit, and argyria has followed the long-continued use of the former drug. Tonics, and especially strychnin are good, and sometimes alcohol, should the patient's ailment not be of alcoholic origin, is of service. It must always be administered with the greatest caution. Change of air and scene, where possible, should be recommended, and a visit to a continental spa, which combines a holiday with a course of mineral waters and a simple and quiet life, sometimes results in prompt improvement.

TOXIC GASTRITIS.

DEFINITION—This name is given to the necrotic and inflammatory changes produced in the stomach by the ingestion of poisonous doses of various substances, *e.g.*, the concentrated acids or alkalies, arsenic, alcohol, phosphorus, corrosive sublimate, &c.

Morbid Anatomy.—The part of the stomach (*e.g.*, the greater curvature towards the cardiac part) with which the poison first comes into contact usually suffers most. The changes vary greatly according to the poison and its degree of concentration. There may be congestion (more or less severe), hæmorrhagic areas, ulcers, sloughs, great softening or complete solution of part of the stomach wall. The affected mucous membrane often shows characteristic appearances, thus it is intensely inflamed after alcohol, phosphorus and many others; pale, corrugated and sodden after carbolic acid; yellowish coloured in places after arsenic or nitric acid; greyish, brown or black after sulphuric or hydrochloric acid. Sulphuric acid is the most frequently used of the poisons, and causes the greatest amount of corrosion. Actual perforation is more common than after any other poison, and occurs in a large number of the cases. Recovery may follow in these toxic cases even after extensive ulceration, and the resulting deformity from cicatricial contraction may be severe, producing hour-glass contraction, stenosis of the pylorus, &c.

Symptoms.—The local symptoms are very severe and sudden, particularly the pain, retching and vomiting, and the general symptoms of collapse and intoxication are in keeping therewith.

Treatment.—Administer an antidote at once, and try to allay inflammation by ice, sedatives, and especially by the

use of morphia (hypodermically) if pain is severe. The sooner a strong acid or alkali, if it has been swallowed, is neutralised the better is the chance of the patient's recovery. An icebag should be continuously applied over the stomach, and the patient's strength maintained by rectal alimentation.

INFECTIVE OR MYCOTIC GASTRITIS.

There are several varieties, according to the form of infection which is present. The best known is *phlegmonous*, in which there is a purulent infiltration of the stomach wall, particularly marked in the submucous coat, either diffusely spread over a large part of the wall or collected within a circumscribed area forming an abscess. The diffuse form is twice as frequent as the abscess. The purulent infiltration may or may not extend throughout the wall of the stomach, penetrating into its cavity or into the peritoneum. The former is the more frequent. It may be likened to a phlegmonous erysipelas in the skin, and it is interesting to note that a streptococcus is the most frequent of the organisms found in those cases which have been examined bacteriologically.

Symptoms.—They are those of very severe gastritis, and pus may be vomited.

Another form of infective gastritis has been called *membranous* because the mucous membrane becomes covered by a coating, forming a sort of membrane of varying thickness, colour and extent. It is comparable to the cases of membranous pharyngitis, and indeed the cases often show membrane in some other part as well, particularly the throat, œsophagus or intestine. The membrane is formed of fibrin, mucus and cells, and has nothing peculiar about it. The names croupous and diphtheritic have been given to it on account of this fibrinous character. It is always

due to some definite bacterium, though not always to the same one. Löfflers bacillus, the pneumococcus, and the streptococcus may be mentioned as the chief of these. It is nearly always secondary and merely exaggerates the symptoms due to the primary disease.

Treatment.—In the *phlegmonous* form little can be done except to relieve symptoms, especially pain and vomiting. In *diphtheritic* cases, antitoxin should be administered in the hope that it may not be too late. In every case where a fungus is present, lavage with a suitable antiseptic is indicated.

ACUTE DILATATION OF STOMACH.

Etiology.—This rare condition has followed upon the rapid swallowing of enormous quantities of food and drink.

Symptoms.—The local symptoms of distress are subordinate to the great general depression and embarrassment of the respiration and circulation. There is great dyspnoea and a rapid soft pulse. Death may quickly supervene unless the stomach be emptied.

Treatment.—Where it is possible to attempt treatment, empty the stomach at once and administer stimulants, such as ether, strychnin, and sometimes strophanthin hypodermically.

CHRONIC DILATATION OF STOMACH.

Etiology.—This comparatively common condition in middle-aged and elderly people may arise from (1) some

obstruction to the emptying of the stomach, *e.g.*, cancer of the pylorus or cicatrisation of an ulcer or pressure upon the duodenum by a new growth of the peritoneum, pancreas, &c.; or (2) weakness of the muscles of the stomach. The most common cause of such weakness is chronic gastric catarrh, when the resulting dilatation may in time be great. Functional disturbances of digestion may also cause weakness, resulting in dilatation, which is generally of moderate extent and of temporary duration.

Symptoms.—There may be no symptoms, particularly in moderate and temporary cases. When present, they vary with the cause and the degree of distension of the organ, and resemble those of *dyspepsia* and *chronic gastric catarrh*, except that *thirst* may be prominent. There is *epigastric discomfort or pain* of a diffuse character, which generally increases in severity until relieved by *vomiting*. This latter is the most characteristic of all the symptoms. It may occur spontaneously or be voluntarily induced at intervals, the vomit consisting of large quantities of fluid, mucus and food of a characteristic sour odour. The quantity varies from about one to six pints or more. The increased acidity is due to organic acids more than to the hydrochloric acid, which is generally diminished, though it may be increased. Microscopically the vomit shows bacteria, yeasts and sarcinae. *Gaseous eructations* are frequent. *Flatulence* generally increases until relieved by vomiting, but occasionally it becomes greater immediately after vomiting. *Tetany* is occasionally observed, particularly in children. The *general nutrition* suffers and great emaciation may result. *Inspection* may discover a great prominence in the epigastric region, or even the outline of the distended stomach, the lesser curvature forming a groove about two inches below the ensiform cartilage or just above the umbilicus, the greater curvature, often not so well seen, passing obliquely from the left below the umbilicus towards

the pubis and then upwards towards the right side. Active peristaltic movements of a spontaneous origin, generally going from left to right, may be seen, or they may be induced by flipping the abdomen with a wet towel or by distending the stomach with tartaric acid and bicarbonate of soda. *Palpation* may elicit the feeling of a hard ball or air cushion when the stomach is distended, or it may feel gas gurgling through the pylorus. Bimanual palpation or shaking the patient often elicits a splashing sound, which is pathognomonic of dilatation when it is obtained two to three hours after eating.

Treatment.—The cause of the dilatation must be first investigated. If there is pyloric stenosis, whether due to a tumor, simple ulcer, or thickening of the pylorus, operative interference should be considered. A gastro-enterostomy often adds months of comfortable existence to a patient whose stricture is malignant and not removable, and is the only adequate measure for the patient's relief. Dilating the pylorus and pylorotomy are operations sometimes warranted.

For gastrectasis, the result of atony of the stomach wall, there is no better method of treatment than repeated lavage, first using an alkaline lotion to dissolve the tenacious mucus, and thereafter a suitable solution of permanganate of potash or other innocuous antiseptic. Lavage carried out in this way is often most successful, and a meal can be taken after the operation with much greater comfort to the patient and with a better chance of its being properly digested. Careful dieting with small-sized meals, which should consist of easily digested constituents and a minimum of fluid, is most important. Where fermentation is present, antiseptics, such as hyposulphite of soda in 10 to 15 grain doses or small doses of carbolic acid, may be administered by the mouth, or the much more efficacious method of washing out the stomach with a solu-

tion of hyposulphite of soda (1 to 2 drachms to the pint) or permanganate of potash (10 grains to the pint) may be employed. Massage over the region of the stomach, and sometimes electrical applications, are found to be helpful, although the introduction of one pole into the stomach demands care on the part of the operator. Tonics, such as strychnin, are often advantageous.

ULCER OF THE STOMACH.

Syn. *Perforating, Round, Simple or Peptic Ulcer.*

Etiology.—The gastric juice does not attack the stomach wall so long as the latter remains healthy, but as soon as any part of the stomach wall becomes sufficiently devitalised, the gastric juice may attack and digest it just as it does the ingested food. An ulcer will result whose depth may extend through any or all of the coats of the stomach wall. The gastric juice is thus a constant agent in the production of the ulcers. It may be normal or abnormal, and when the latter it probably acts more powerfully. The other agents in the causation of the ulcer, viz., those which bring about devitalisation, are only partially, if at all, known, and almost every one theoretically possible has been advocated. Thus the arteries, veins, nerves, constitutional states and direct irritants have all been blamed, but the requisite proof is wanting in all of them. The oldest theory is the embolic one, and it is theoretically a very good one, for if an embolus were to obstruct a branch of any of the gastric arteries, the part of the stomach wall supplied by it would become necrosed and fall an easy prey to the gastric juice. The infarcted area would result in an ulcer. Experience has, however, been able to bring forward very

little proof in its support, for not only have emboli, or even the possible sources of emboli, *e.g.*, cardiac vegetations, rarely been found in cases of gastric ulcer, but it has been shown experimentally that when numerous emboli are present in the circulating blood they rarely enter the arteries of the stomach. Thrombosis would be an efficient cause but it is equally rarely seen. Vasomotor spasm was accordingly suggested as a possible cause of an anæmia of one or more parts of the stomach wall sufficiently severe to interfere with its healthy nutrition and to allow of gastric digestion and ulceration following, but there is neither any proof that such local vasomotor spasm occurs nor any satisfactory reason why it should. On the side of the veins, it has been pointed out that great venous engorgement may lead to stasis and devitalisation of one or more parts of the stomach wall, and a gastric ulcer certainly occurs occasionally in cases of cirrhosis of the liver, but most cases of gastric ulcer show no venous engorgement or any reason why there should be venous engorgement. A perverted trophic nerve (vagal) influence, with or without vasomotor spasm, might have some influence if it could be shown to exist. Constitutional states, on the other hand, are supported by a certain amount of evidence, such as the great frequency of gastric ulcer in chlorotic girls, but they can hardly explain all cases, as in many cases of gastric ulcer there is neither chlorosis nor any other constitutional derangement. Direct irritants, such as chemical poisons or external blows, may conceivably damage the gastric mucous membrane, as may also irritating substances of any kind, such as ingested solid bodies or abnormal products of digestion, and the solvent action of the gastric juice may do the rest. There is no doubt that chemical poisons introduced from without may cause ulceration of the stomach, but external blows or ingested solid bodies, however irritating they may be, probably do little damage. It is more difficult

to say how much influence irritating digestive substances have. They are a fruitful source of catarrh, and gastric ulcer often has a history of pre-existing catarrh; it is, moreover, reasonable to think that at least some long-standing catarrhs may so deteriorate the mucous membrane that in time it may be eroded by the gastric juice and an ulcer result. The causes at work in the production of gastric ulcer are thus in all probability not always the same. Different causes or different combinations of causes may be operative in different cases to produce the requisite condition of deterioration of the mucous membrane. Hence the importance of avoiding or checking all such irritations before they reach this effective stage.

Morbid Anatomy.—The stomach is its most common site, but it is not infrequent in the duodenum (above the biliary orifice, very rarely below it), and it is rare in the lower end of the œsophagus. The duodenal ulcer is much more common in men, while the gastric ulcer is somewhat more common in women, until after middle life, when it is about equal in both sexes. The commonest site for the gastric ulcer is the pyloric region of the stomach on the posterior surface, near the lesser curvature, but it may be present on the greater curvature or anterior surface, or, indeed, anywhere in the stomach. There is usually only one, though occasionally there are two or three, or even many. The size varies, being smaller in recent cases than in long-standing ones. The average is somewhere about half-an-inch in diameter, though some, particularly very old ones, may measure several inches. The margins are well defined, being often sharp and clean as if cut with a knife. The depth varies, the floor being usually formed by part of the circular muscular coat. Both margins and floor are smooth, and the former often present a terraced appearance owing to the gap in the mucous membrane being greater than that in each of the succeeding or deeper coats, an

appearance such as would be obtained if a punch were used in producing the ulcer, a smaller instrument being used for the submucous than for the mucous coat, and a still smaller one for the muscular than for the submucous. This terraced appearance is best seen in so-called acute ulcers, as in those of very long standing the margins are smooth, rounded and thickened. These often show much cicatrization and contraction, producing various deformities. The most striking of these is the hour-glass contraction of the stomach, which is produced by a belt or girdle-like ulcer in its body, but the most important is the dilatation of the body of the stomach produced by a cicatrising ulcer at the pylorus. The deeper ulcers may perforate the wall of the stomach and allow its contents to escape into the peritoneal cavity. This is most likely to occur after a meal, and if the escaped contents be large, death from shock may rapidly ensue, so rapidly in some cases, particularly if the ulcer be unsuspected, as to suggest an irritant poisoning. This free escape of the gastric contents into the peritoneal cavity is more likely to occur when the ulcer is on the anterior surface, and, if the patient survive the shock, an acute peritonitis of more or less general distribution will be set up. On the other hand, when the perforation is on the posterior surface of the stomach the escape of the gastric contents is less free, and adhesions generally form and limit the area of inflammation. The perforation itself may be dangerous, as an important artery (splenic or branch of coronary) or vein may be eaten into and a serious or fatal hæmorrhage ensue, or the pleura, pericardium or even the heart (very rare) may be perforated, or a gastro-colic or gastro-duodenal fistula may be established, but usually a restricted area of inflammation (simple or suppurative) results. Abscesses in or around the pancreas, liver or spleen may thus arise. Many of these are bounded above by the diaphragm, constituting what is now known as a subphrenic abscess, or, if gas be present, as a subphrenic

pyopneumothorax. These subphrenic abscesses are most frequently situated in the region of the lesser omental sac, and anterior to the left lobe of the liver, not extending to the right of the suspensory ligament of the liver, except in the case of duodenal ulcers, when they often do so. They contain pus, usually of a watery consistence, often mixed with stomach contents and also, particularly if the perforation be recent, with gas or air. The air, if not renewed, may be quickly absorbed, but the pus may remain for long periods without spreading beyond its early boundaries. There is always the risk, however, of metastatic abscesses forming in the liver, lungs, or other situation, and general peritonitis, pleurisy or pericarditis may follow at any time.

Symptoms.—Of the three leading symptoms, viz., *signs of indigestion, pain, and hæmatemesis*, the last is the most striking and characteristic. It is true that it is subject to great variations as to frequency of occurrence and recurrence, as to the amount of blood vomited, and also even as to the appearances it presents and the effect it has upon the patient; but in a typical case these are all so characteristic as to leave no room for doubt in the diagnosis. The amount of blood is considerable—several ounces to a pint or more. It is bright red in colour and fluid when vomited, clotting afterwards as normal blood does when shed. Less commonly it is brought up in a partly clotted state, but when it is allowed to remain for some time in the stomach it gets mixed with the food and altered by the gastric juice to a dark brown grumous fluid, not unlike coffee grounds. It may recur once or oftener within a day or two, or at longer intervals. It rarely causes death directly, but it may cause collapse or fainting, and ultimately it is liable to be followed by anæmia, proportionate in degree to the blood loss. In exceptional cases there is no hæmatemesis, though there is escape of blood, it may be in large amount, into the stomach, because the blood passes through the pylorus

into the duodenum and is ultimately passed in the stools as a black tarry substance, known clinically as *melæna*. While the vomit in cases of gastric ulcer does not always contain blood, it does always contain an excess of hydrochloric acid. In a certain proportion (about a third) of all cases of gastric ulcer there is neither hæmatemesis nor *melæna*, when attention must be paid to the other symptoms. *Pain* is the most frequent of them, and though, like the hæmatemesis, it shows great variation in frequency, recurrence and degree of severity, yet in typical cases it is intense and paroxysmal, coming on at varying intervals after taking food. It is frequently localised to the epigastrium, but often radiates to the back and to the sides. It may recur after every meal, sometimes only a few minutes afterwards, but at others after longer intervals up to an hour or more. Some writers have claimed on insufficient evidence that this interval of time indicates the site of the ulcer, the shorter the period the nearer to the cardiac end, and the longer the nearer to the pylorus. It is usual, even in bad cases, for the pain to become slight or disappear altogether for considerable intervals—weeks or months. It is also liable to become exaggerated by various outside influences, such as *exposure to cold*, mental or physical excitement, or by menstruation. In exceptional cases it is absent altogether, or when present it may have no relation to the taking of food. The effect of *pressure* deserves special attention. Slight pressure nearly always causes discomfort. A waist-belt or stays, or even contact with the bed-clothes, may be unpleasant or even unbearable. Deep pressure, on the other hand, while it usually increases the pain, may relieve it. The other symptoms of gastric ulcer are much less definite. They may be grouped under the heading of indigestion or dyspepsia. Usually there is a history of dyspepsia of varying degree of severity but of considerable duration. It is probably due in most cases, if not in all, not to the gastric ulcer itself, but to a preceding or accom-

panying chronic gastritis, or to a nervous, blood or other constitutional derangement, or some combination of them. Some cases of gastric ulcer, on the other hand, remain latent, giving rise to no symptoms. A sudden hæmatemesis or signs of perforation may reveal them. Palpation may detect the thickening which occurs around some old ulcers.

Diagnosis.—This is easy and certain in typical cases, while in others it can only be tentatively made.

Prognosis.—It is very good. Most uncomplicated cases get well. It is, however, a trouble needing prolonged care and attention. In hospital, cases generally steadily and quickly progress towards recovery, whereas they often relapse after returning to their own homes, merely because they are not under the same continual supervision. There may be one or more periods of recurrence of symptoms before ultimate recovery is assured. Death may supervene within a short or long period after the recognised onset of the disease from hæmorrhage, perforation or other complication; but surgical aid has become available in many such serious cases, and a gastro-jejunostomy or other operation may grant a new lease of life to cases apparently hopeless.

Treatment.—Much depends on the symptoms present. If the ulcer is active, and especially if there is hæmorrhage from it, absolute rest in bed and rest for the stomach are imperative. The patient can be kept alive by means of rectal alimentation. Ice can be given to suck, while an ice-bag placed over the stomach soothes pain and arrests any tendency to peristalsis. Morphia ($\frac{1}{4}$ to $\frac{1}{2}$ grain) and ergotin (2 grains) hypodermically are of service, but where there is severe and repeated hæmorrhage, operative interference should be entertained. In cases of perforation the aid of the surgeon is imperative. In most instances of gastric ulcer, however, dieting is alone called for. The

food should be of a kind which is easily digested, given in small doses, and hyperacidity, so constantly present, should be combated, either by giving an alkali *after* food or a small dose of the dilute hydrochloric acid (5 to 10 minims) immediately *before* food. Pain may be soothed by the use of an alkali, often combined with dilute hydrocyanic acid (3 minims). Lavage is only necessary where there is gastric dilatation. Anæmia, often present in young girls the subjects of gastric ulcer, should be treated with iron, and preferably with a proto-salt.

DUODENAL ULCER.

This ulcer is most common in the first part of the duodenum. It occurs with comparative infrequency in the second part, and rarely in the third part, below the biliary orifice. It presents much the same characters as does the gastric ulcer (which is about thirty times as frequent), but differs from it in certain respects. It is far more common in men than women, being most frequent in middle-aged men, though it is not infrequent in children, and a certain number (much smaller now than formerly, when antiseptic treatment of wounds was not so complete) occurs after extensive cutaneous burns, caused by, it is thought, septic emboli reaching the duodenal mucosa and producing hæmorrhagic infarcts. Duodenal ulcers tend to perforate more frequently than do gastric ulcers. They are more often latent, and symptoms, when present, while they resemble those of gastric ulcer, are less definite. Vomiting and hæmatemesis is less frequent, and melæna more frequent.

Treatment.—It is doubtful whether operative interference is not even more frequently indicated in duodenal than

in gastric ulcers. Medical treatment consists in careful dieting, often preceded by a period during which rectal alimentation is alone used, but there is little to be said in addition to the measures already suggested for gastric ulcer.

CANCER OF THE STOMACH.

Etiology.—This common and important disease is almost always primary, but it is not possible in the light of our present knowledge of the general etiology of cancer to say much about its causation in the stomach. Like cancer elsewhere it increases as age advances, so that the majority of cases occur after sixty. It occurs very exceptionally in quite young children. It is slightly more common in men than women, but it cannot at present be said that prolonged irritation or any other likely agent, such as gastric ulcer or other pre-existing disease, has been shown to have any predisposing influence.

Morbid Anatomy.—The pyloric region is the most frequent site of all the forms of gastric cancer, and it is curious to note how very rarely it passes beyond the pylorus into the duodenum. Its next most frequent site is the lesser curvature, next comes the cardiac end, and, while the entire stomach is often involved, the posterior wall and other parts are less frequently affected. The appearances of the growths vary much, both macroscopically and microscopically, and it is customary to arrange them in the usual groups adopted in the classification of cancer, viz., medullary or encephaloid, scirrhus, colloid, cylindric or adeno-carcinomata or columnar-celled epitheliomata and squamous epitheliomata. They all, except the last, take their origin

in the glandular epithelium of the mucous membrane of the stomach, whose overgrowth constitutes the essential part of the tumor, but among the epithelial cells there is a varying amount of fibrous tissue stroma produced by the fibrous tissues of the stomach through the irritation caused by the epithelial overgrowth. When the epithelium in the tumor preserves its cylindric shape and its tubular arrangement it is called cylindric or adeno-carcinoma; when, on the other hand, it quickly loses both, and the cells, now round, flattened or polymorphic, come to lie in spaces or alveoli within a fibrous matrix, one of the other terms is used. If the alveoli be large and the cells be numerous relative to the amount of stroma, the cancer is called medullary, whereas if the reverse be the case it is called scirrhus. This distinction is only useful in a broad way, for tumors and even the same tumor in different parts may present the characters of both. There are often, too, insensible gradations between the cylindric and medullary arrangements, perfect tubes being seen in one part and large spaces containing rounded and polymorphic cells, showing no attachment to their walls, in another; while intermediate parts show that the latter has been produced by the former through proliferation and desquamation of the lining columnar cells. This has led some writers to assert that all cancers of the stomach are really adeno-carcinomata, but whether this is true or not, and it probably is not, is of no practical importance. Cancers begin in the mucosa by one or more of the epithelial cells taking on cancerous characters, and they may keep for a time or lose at once their normal cylindric shape and tubular arrangement. Colloid cancers are those which show much colloid or mucoid change within the tumors. The squamous-celled cancer of the stomach originates in the epithelium of the œsophagus, and thus occurs exclusively at the cardiac end, though it may involve the whole of it. It is rare. These distinctions are

based upon characters chiefly recognisable by the microscope; but they are not the only ones upon which the classification depends. Thus the medullary and many of the adeno-carcinomata form large, greyish-white, soft fungoid-like tumors projecting into the lumen of the stomach and extensively invading its walls. The surface is irregular, polypoid or cauliflower-like, and often ulcerated more or less deeply in the centre. The medullary forms are of quick growth, quicker in most cases than the cylindric varieties. The scirrhus cancer, on the other hand, is of slower growth and forms a hard, dense thickening, infiltrating the stomach walls rather than projecting as a tumor into its cavity. Its inner surface may be smooth, but is usually rough and ulcerated but not projecting much, if any, above the neighbouring healthy mucous membrane except at its margins, which are prominent. It leads to much cicatrisation, and since it is, like the medullary form at any rate, most frequently situated in the pyloric region, it may cause much pyloric stenosis, usually, though not always, followed by dilatation of the stomach. Some adeno-carcinomata form malignant ulcers with so little elevation of their edges and so little thickening and induration of their floors that they cannot be detected by palpation through the abdominal wall, or even after a laparotomy through the wall of the stomach itself. The stomach may require to be opened or the ulcer examined microscopically before their true nature is discovered. Scirrhus cancer sometimes invades a large or the greater part of the stomach wall, usually extending upwards from the pylorus as a uniform thickening, up to half-an-inch or more, of its coats, particularly its sub-mucous and mucous, converting them into a hard, fibrous substance mostly consisting of fibrous tissue, the cancer cells being very few in number and detectable only in places. The mucous membrane may be rugose but apparently normal, though the lumen of the stomach is

greatly diminished. It is probable that many cases of cirrhosis of the stomach are of this nature.

Whatever be the variety of cancer, it tends to extend beyond the stomach and invade other organs, both by direct extension and by metastases. The lymphatic glands around the stomach are most frequently involved. They become enlarged and matted together. Other glands at a distance may be become affected, *e.g.*, the thoracic, inguinal and supra-clavicular. The liver becomes invaded by multiple secondary nodules in about 75 per cent. of all cases. The peritoneum is frequently and may be rapidly invaded, the parietal peritoneum being studded with innumerable small or varying-sized nodules, and the visceral peritoneum, particularly the omentum, similarly affected, the latter being often rolled up into a solid mass lying transversely across the abdominal cavity. In colloid cancers the whole peritoneal cavity may be filled with colloid material like jelly, as if it had been poured in a liquid form into the peritoneum and had congealed afterwards. Other abdominal organs, such as the pancreas, colon (a gastro-colic fistula may result) and spleen may be invaded by gastric cancer, and not infrequently subcutaneous secondary nodules appear in the abdominal wall. Metastasis may also occur in the pleura, lungs, brain and other parts.

Symptoms.—The onset is insidious. Dyspeptic symptoms may appear and gradually become more and more marked, but they are often absent. *Loss of appetite* is one of the earliest of the important symptoms. It is almost always present and generally gets steadily worse. *Nausea* is also frequent. *Progressive loss of strength, anæmia* and *emaciation* become noticeable, sometimes at an early period, and may be the only symptoms present for a long time. *Vomiting* is an important and very frequent symptom. It may come on early or not

until other symptoms have been present for some time. It is usually absent when the cancer is situated at the lesser curvature or in diffuse scirrhus infiltration of the body of the organ. It is usually infrequent at first, but its occurrence gradually increases as the case progresses, until it may recur several times a day. It is, nevertheless, subject to considerable variation, and in the last stages, where there is much dilatation, it may almost or completely cease. The vomit usually consists of partially-digested food (some of which may have been ingested days or even weeks previously) and mucus in a greyish or dark fluid having an offensive smell. *Blood* is frequently present, rarely in considerable quantity, and unaltered, as in gastric ulcer, but usually having the "coffee-ground" appearance. It may be so slight in amount that its presence is not revealed until the spectroscope or the microscope is used. Small fragments of the tumor occasionally, though rarely, appear in the vomit, in which the microscope can detect the structure characteristic of cancer. A more important feature of the vomit is the fact that in the majority of cases it contains *no free hydrochloric acid*, though it often contains *free lactic acid*. *Pain* is the most constant symptom of all. Exceptionally it is slight or absent. Usually it is severe, of a burning or gnawing character, and, while fairly constant, it is generally aggravated by food and seldom much relieved by vomiting. Pressure, both slight and severe, increase it, though it is not so localised and is rarely so acute as in simple gastric ulcer.

As the anæmia (it is typically a secondary anæmia, though some cases may resemble the pernicious form) advances, dropsical symptoms, dependent thereon, supervene. Œdema of the ankles and legs is common, and thrombosis of the femoral vein sometimes occurs. Mild fever, up to about 101°F., is often observed, and is generally thought to be caused by some toxin produced by the growth or by some accompanying inflammatory condition

of the stomach or peritoneum. The emaciation and loss of strength (the muscular weakness is often striking) steadily increase, and the resulting general condition of ill-health ultimately constitutes an appearance (the pallid, slightly-jaundiced skin frequently showing brown spots, the wasted and exhausted body) to which the name *malignant cachexia* is given.

PHYSICAL SIGNS.—Palpation may detect a tumor, but it is important to remember that about two-thirds of the stomach lies beyond the reach of palpation, behind the ribs. Pyloric tumors are usually firm, hard, nodular and painful on pressure. They are most commonly felt in the epigastric or umbilical regions, more to the right than to the left of the middle line. Exceptionally they may be felt lower down towards the iliac region, and occasionally they are so high up beneath the liver and chest wall that they cannot be felt at all. Tumors of a part of the anterior wall and of the greater curvature can also be felt, usually in the epigastric region or lower, but tumors of the cardiac end cannot be felt, nor can those of the fundus, the posterior wall, or the lesser curvature, except occasionally when they are very large. Valuable corroborative evidence may be obtained by finding that the liver is enlarged or nodular, and that the peritoneum or lymphatic glands are similarly affected.

Diagnosis.—Loss of health and strength in an aged person, of insidious onset and unexplained cause, should always suggest cancer. Loss of appetite and nausea with some gastric discomfort are also strongly suggestive. In most cases, however, the diagnosis can only be made tentatively for some considerable time. When vomiting (with the presence of blood and the absence of hydrochloric acid in the vomit) and pain also appear it becomes more probable, and is rendered certain by the detection of a tumor. The diseases from which it is

most difficult to distinguish it are—simple gastric ulcer, pernicious anæmia and chronic gastritis in the aged. In simple gastric ulcer the pain is more severe, more spasmodic, more localised, and more relieved by vomiting; the hæmatemesis is greater in amount, less frequent, and consists generally of unaltered blood; the vomit contains excess of free hydrochloric acid; the emaciation is often slight; the patient is generally younger, and a tumor is rarely present. In pernicious anæmia the characters of the blood are not those of a secondary anæmia, *vide* pages 355 and 358. In chronic gastritis pain and vomiting are less frequent and severe, hæmatemesis is rare, free hydrochloric acid is present except in long-standing cases with atrophy of the mucosa, and in them the duration of the illness itself may suffice to exclude cancer.

Prognosis.—The patient steadily progresses in a downward direction. Short periods of improvement may at times occur, but the onward march is soon resumed and death usually occurs within a year or a little more after the onset of the symptoms. In some cases it is less and in others, particularly the diffuse scirrhus forms, it may extend to about two years. Persistent hiccough is a bad sign, as it often comes on shortly before death. Asthenia is the common method of death, which is often hastened by some complication.

Treatment.—The treatment can only be palliative in most cases. We have occasionally seen cases in which a pyloric tumor can be completely removed by pylorotomy. Lavage is advantageous where there is dilatation with the presence of fermentative organisms. Sedatives, such as bismuth subnitrate (10 to 30 grains), cerium oxalate and morphia are often necessary. Where there is pyloric stenosis a gastro-enterostomy or gastro-duodenostomy should be performed.

DIAGNOSTIC TABLE of Gastric Cancer, Gastric Ulcer, and Chronic Catarrhal Gastritis.

<p>GASTRIC CANCER.</p> <hr/> <p>1. Tumor is present in three-fourths of the cases.</p> <p>2. Rare under forty years of age.</p> <p>3. Average duration about one year, rarely over two years.</p> <p>4. Gastric hæmorrhage frequent, but rarely profuse; most common in cachectic stage.</p> <p>5. Vomiting often has the peculiarities of that of dilatation of the stomach.</p> <p>6. Free hydrochloric acid usually absent from the gastric contents in cancerous dilatation of the stomach.</p> <p>7. Cancerous fragments may be found in the washings from the stomach or in the vomit (rare).</p>	<p>GASTRIC ULCER.</p> <hr/> <p>1. Tumor rare.</p> <p>2. May occur at any age after childhood. Over one half of the cases under forty years of age.</p> <p>3. Duration indefinite; may be for several years.</p> <p>4. Gastric hæmorrhage less frequent than in cancer, but oftener profuse; not uncommon when the general health is but little impaired.</p> <p>5. Vomiting rarely referable to dilatation of the stomach, and then only in late stage of the disease.</p> <p>6. Free hydrochloric acid usually present in the gastric contents.</p> <p>7. Absent.</p>	<p>CHRONIC CATARRHAL GASTRITIS.</p> <hr/> <p>1. No tumor.</p> <p>2. May occur at any age.</p> <p>3. Duration indefinite.</p> <p>4. Gastric hæmorrhage rare.</p> <p>5. Vomiting may or may not be present.</p> <p>6. Free hydrochloric acid may be present or absent.</p> <p>7. Absent.</p>
---	--	--

DIAGNOSTIC TABLE—Continued.

GASTRIC CANCER.	GASTRIC ULCER.	CHRONIC CATARRHAL GASTRITIS.
<p>8. Secondary cancers may be recognised in the liver, the peritoneum, the lymphatic glands, and rarely in other parts of the body.</p>	<p>8. Absent.</p>	<p>8. Absent.</p>
<p>9. Loss of flesh and strength, and development of cachexia usually more marked and more rapid than in ulcer or in gastritis and less explainable by the gastric symptoms.</p>	<p>9. Cachectic appearance usually less marked and of later occurrence than in cancer, and more manifestly dependent upon the gastric disorders.</p>	<p>9. When uncomplicated, usually no appearance of cachexia.</p>
<p>10. Epigastric pain is often more continuous, less dependent on taking food, less relieved by vomiting, and less localised than in ulcer.</p>	<p>10 Pain is often more paroxysmal, more influenced by taking food, oftener relieved by vomiting, and more sharply localised than in cancer.</p>	<p>10. The pain or distress induced by taking food is usually less severe than in cancer or ulcer. Fixed point of tenderness usually absent.</p>
<p>11. Causation not known</p>	<p>11 Causation not known.</p>	<p>11. Often referable to some known cause such as abuse of alcohol, gonorrhoea, and certain diseases, as phthisis, Bright's disease, cirrhosis of the liver, &c</p>
<p>12. No improvement or only temporary improvement in the course of the disease.</p>	<p>12. Sometimes a history of one or more previous similar attacks. The course may be irregular and intermittent. Usually marked improvement by regulation of diet.</p>	<p>12. May be a history of previous similar attacks. More amenable to regulation of diet than is cancer. —(Osler, quoted from Welch.)</p>

TABLE showing the Connection between certain Symptoms of Diseases of the Stomach and of other Organs.

Gastralgia. Cardialgia. Gastrodynia. Pain.	Pain is a marked symptom of diseases of the stomach, but gastric pain may be due to organic or functional disease of the colon, to <i>aneurism of the abdominal aorta</i> , and <i>incipient disease of the vertebrae</i> . Care in diagnosis is therefore required. Pain may also accompany emptiness of the stomach, relieved by the ingestion of a small quantity of food. Pain after taking food may be due to organic causes, ulcer, cancer, or functional in hysterical persons and those of weak digestions. For the diagnosis of ulcer and cancer, see the account of these diseases.
Alteration of Appetite. <i>Anorexia</i> —Loss of Appetite.	Loss of appetite may be due to loss of digestive power, or to organic disease. Loss of appetite is common in fevers and most morbid states not necessarily connected with the digestive organs.
<i>Bulimia</i> — Insatiable Hunger.	<i>Bulimia</i> (<i>Bovs</i> "an ox," and <i>λιμος</i> "hunger," or <i>βο</i> and <i>λιμος</i>). Excessive hunger may be due to worms, diabetes, and disease of the mesenteric glands. It may also accompany one of the forms of gastric pain.
<i>Pica</i> — Depraved Appetite.	Depraved appetite for plaster and for dirt of all kinds is seen in some children, and in hysterical and pregnant women.
Merycismus.	A power which some persons have of regurgitating their food and chewing the cud. The condition occurs most frequently among hysterical women, epileptics, &c.
Regurgitation.	May occur in the forms of eructations or belching of gas or small quantities of food. Pyrosis or water-brash is a form of eructation, accompanied with the discharge of a quantity of acid, alkaline or neutral fluid from the stomach. The disease is more common in Scotland than in England. Some consider pyrosis as a result of a catarrhal affection of the mucous membrane of the stomach, accompanied with hypersecretion of gastric fluid; others to excessive secretion of saliva, swallowed and arrested at the cardiac end of the œsophagus and then regurgitated.

TABLE — Continued.

Vomiting. Emesis.	<p>Common in many diseases, and in some forms of dyspepsia preceded by nausea (<i>naus</i> "a ship"—sea sickness). Cancer and ulcer of the stomach. Pregnancy, and among hysterical women. Is an early symptom of scarlet fever and small pox. Has been known in the <i>incipient</i> stage of <i>phthisis</i>, in <i>Addison's disease</i>, <i>Bright's disease</i>, and in <i>cerebral diseases</i>. Care must therefore be taken in referring vomiting to its proper source.</p>
Peristaltic Unrest.	<p>This is a most distressing symptom, coming on after eating, in which the peristaltic movements of the stomach and intestines are increased, accompanied with borborygmi and gurgling, which, as they can be heard at a distance, are most distressing to the sufferer. Sometimes there is anti peristalsis, when food, &c., may be regurgitated through the mouth.</p>
Palpitation. Cough Singultus (Hiccough)	<p>Palpitation of the heart, cough, and hic cough may all accompany gastric disorders. I have seen troublesome hiccough during an attack of gouty dyspepsia.</p>
Hæmatemesis.	<p>This may occur in certain <i>general diseases</i>—puapura, scurvy, cirrhosis of the liver, acute yellow atrophy of the liver, diseases of the spleen, &c. It may occur as a form of vicarious menstruation. It is chiefly in simple ulcer and in cancer of the stomach that hæmatemesis occurs. See the account of both these affections. It must be remembered that the stomach may contain a large quantity of blood, accompanied by all the signs of hæmorrhage, and yet hæmatemesis be not present. Suspect this when there is pallor on the face, dimness of vision, giddiness, or even fainting. In <i>simple congestion</i> of the stomach, dependent in most cases on latent cirrhosis of the liver, hæmatemesis may occur.</p>

VI.—DISEASES OF THE INTESTINE.

MALFORMATIONS AND DISPLACEMENTS.

Malformations and displacements of the intestine are of practical importance to the physician, since some of them may cause intestinal obstruction, and they are most conveniently referred to under that heading.

INTESTINAL OBSTRUCTION.

Etiology.—The intestine may be obstructed by changes in its contents or in its walls. Its natural contents—the fæces—may become impacted, or abnormal contents, such as articles of food, gall stones, foreign bodies which have been swallowed, intestinal worms, &c., may block its lumen. These are all of infrequent occurrence compared with changes in its walls, *e.g.*, hernia, diverticula, strangulation, intussusception, volvulus, strictures, pressure of tumors, &c., some of which are fairly common. Hernia and diverticula are described in surgical text-books, and the others need only short descriptions here.

Strangulation is the cause of fully one-third of all cases of acute intestinal obstruction. Fibrous bands produced by previous peritonitis or an adherent Meckel's diverticulum are the means by which the strangulation is most frequently brought about. Portions of the intestine pass through the loops thus formed and by their subsequent distension, or kinking, strangulation ensues. An adherent vermiform appendix sometimes forms a similar band, and slits in the omentum or mesentery may act in the same way.

Intussusception is almost as frequent a cause of intestinal obstruction as is strangulation. It is constituted by the

entrance of one part of the intestine into another, almost always an upper part into a lower, ileum into ileum, or colon into colon, or (commonest of all) the ileum into the cæcum, carrying in most cases the ileo-cæcal valve with it. It may enter for a distance of about an inch to a foot or more, and in rare cases of the ileo-cæcal variety the the ileo-cæcal valve has reached even the lower part of the rectum. The condition is most common in early life, fully one-third of all cases occurring during the first year, and about two-thirds during the first ten years of life. It is caused by irregularity in peristalsis, which is most active in early life, and many cases have a history of diarrhoea or obstinate constipation, conditions conducive to irregular peristalsis. The method of formation of an intussusception can easily be followed by causing the finger of a glove somewhere below its end to pass into itself. It forms a cylindrical tumor consisting of three layers—an outer or receiving layer (called by Rokitansky the *intussusciptiens*) or sheath, a middle or returning layer, and an inner or entering layer. The latter two together are known as the *intussusception*. Its apex, *i.e.*, its lowest part, is the only fixed point, since its body tends to increase at the expense of the *intussusciptiens*. The intussusceptum rarely slips out spontaneously. It usually goes on entering further and further, and, moreover, it does not enter alone, for it carries with it its mesentery. The pressure to which the invaginated gut with its mesentery is thus subjected interferes with the circulation of the blood. It first obstructs the venous return and then the arterial flow, and the consequent swelling, engorgement and inflammation cause a hæmorrhagic discharge from the bowel (the stools being likened to red-currant jelly) and obstruction of its lumen. The invaginated gut with its mesentery (the intussusceptum) shows the phenomena characteristic of *strangulation*. The passive congestion, loss of nourishment, and oedema pass on to inflammation and gangrene. The intussusceptum

may thus in whole or part slough off, and by extension of the inflammation a general peritonitis is set up. This is the usual course taken by the disease, though in some cases a more favourable issue ensues when the general peritoneal cavity is sealed off by an inflammation of the serous coats of the upper rim of the sheath and of the adjacent healthy intestine and the disease subsides with more or less perfect restoration of the lumen of the bowel. The appearances of the intussusception vary according to its duration. A curved sausage-shaped tumor is formed in all cases. The swelling and congestion are slight to begin with, and even in this early stage, where there is little or no inflammation of the affected part of the bowel or adjacent peritoneum, death may take place from shock. After a few days the inflammation becomes so pronounced that the intussusception is no longer reducible and the tumor soon takes on a deep red or reddish-black colour. This is the appearance usually seen in advanced and fatal cases.

Volvulus.—This condition is not half so frequent as either intussusception or strangulation. It is most common in males between thirty and forty years of age. The sigmoid flexure is the commonest site, the cæcum coming next. An unusually long mesocolon and powerful peristalsis, are usually suggested as causes. The loop of bowel is mostly twisted upon its long axis, a combined twist and kink which causes congestion, swelling, inflammation, gangrene and obstruction, usually ending fatally.

Strictures and tumors rarely cause acute obstruction, but are common causes of the chronic form. Congenital stricture is rare. Acquired stricture is much more common. It most frequently follows the ulceration caused by cancer, tubercle or syphilis, more rarely by dysentery, and still more rarely by typhoid. Of tumors growing within the wall of the bowel, the columnar-celled epithelioma is

the commonest. It may cause obstruction either by ulceration and cicatrisation or by its actual mass. The flexures of the large bowel, viz., cæcal, hepatic, splenic, sigmoid and rectal, are its most frequent sites. Among simple tumors, which comparatively rarely cause obstruction, the papillomata and adenomata are most frequently met with.

Symptoms of Acute Obstruction.—*Vomiting* is the most important symptom. It is at first gastric, then bilious, then stercoraceous. *Pain* usually sets in earlier than the vomiting, spasmodic and colicky at first, it soon becomes continuous. *Constipation* is another prominent symptom, though it is often marked by a pseudo-diarrhœa, i.e., a discharge from the inflamed bowel below the seat of obstruction. The abdomen becomes much distended and tympanitic in most cases because the lower bowel (colon) is the most frequent site of obstruction. The tympanitis may become extreme. It is always present in some degree, except when the site of obstruction is high up in the small intestine. The constitutional symptoms are severe. The face becomes pinched, pale and anxious-looking, and the skin covered with cold clammy sweat. The pulse is rapid and feeble, the thirst great, and the urine scanty and high-coloured and occasionally suppressed, particularly when the site of obstruction is high up in the intestine. Death may occur from shock, collapse or coma, and is rarely delayed more than a week.

Symptoms of Chronic Obstruction.—Habitual constipation of gradual onset is perhaps the commonest symptom. Transient attacks of complete obstruction are common, and it is astonishing how tolerant the system becomes in many cases to the retention of the fæces for long periods—weeks or even months. Vomiting and pain are consequently not so frequent as the experience of acute obstruction would lead

us to expect. They are present in some cases, however, and the vomiting may even be stercoraceous. Alternating attacks of constipation and diarrhoea may be present, and the stools may be hard and scybalous. Similar hard masses may be felt, through the abdominal wall, in the bowels when they seem to be acting pretty regularly. The diarrhoea is not infrequently due to the faecal masses becoming channelled and allowing the contents of the upper part of the bowel to pass through. In later stages the abdomen may become distended, particularly during the times of complete constipation, and active peristalsis of the intestinal coils may be observed through the abdominal wall. The symptoms may last for years, but sooner or later the patient gradually becomes anæmic, emaciated and exhausted, the obstruction becomes acute and ends in death.

Diagnosis.—It is easy in some cases and difficult in others. A careful examination of the history, symptoms, inspection, palpation and percussion of the abdomen, combined with rectal and vaginal examination, will usually enable the physician to be fairly sure. The rectum and colon may be inflated by the injection of fluid or air, or by the use of bicarbonate of soda and tartaric acid, and this may help the diagnosis.

Treatment.—The treatment falls naturally under two divisions—that of *Acute Obstruction* and of *Chronic Obstruction*, but it must not be forgotten that a case of *chronic obstruction* may terminate *acutely*.

For *Acute Obstruction* apply ice locally, and administer sufficient opium hypodermically or by the bowel to alleviate pain until surgical assistance can be obtained. Do not administer purgatives. Enemata may be given for the purpose of clearing out the bowel below the stricture, in the hope that the element of spasm to which the obstruction is often largely due may thereby be removed. The

enemata should, as a rule, be of large size, and may be preceded by an injection of 10 to 15 ounces of warm olive oil. Abdominal distension, which often causes great suffering and interference with the movement of the diaphragm, may sometimes be relieved in one of two ways—either by passing a long soft tube into the bowel per rectum, or by tapping distended coils with a needle through the abdominal wall. For the persistent vomiting nothing affords so much benefit as washing out the stomach. Intussusception and sometimes volvulus may be reduced by inflating the bowel with air per rectum or by large injections of water; but this treatment is more likely to succeed in young children and must be carried out very promptly, otherwise failure is inevitable, and continued attempts are not free from danger. For such cases operation gives the only chance of saving life.

The treatment of *Chronic Obstruction* varies with the cause of the condition. There is often fæcal accumulation, sometimes alone but more often with tubercular, malignant, or other stricture of the bowel. Do not purge, but apply heat to the abdomen; relieve pain by a small dose of opium, often combined with belladonna, and administer first 10 to 20 ounces of olive oil, with sometimes a $\frac{1}{2}$ ounce of turpentine, and half an hour afterwards a large injection of soap and water. These enemata may need to be repeated several times, and if relief is not obtained and no flatus pass the stricture, surgical assistance should be at once procured. Of course malignant and tubercular strictures may be inoperable, but opening the abdomen gives the patient the only chance of relief. Salol and charcoal and other intestinal antiseptics are frequently advantageous where accumulation of gas is largely responsible for the pain.

DEGENERATIONS OF THE INTESTINES.

ATROPHY.

Atrophy follows prolonged catarrh of the bowel and also obstruction to its lumen. When the bowel above the seat of obstruction becomes dilated, it causes thinning of the mucous membrane and loss of glandular tissue.

HYPERTROPHY.

Hypertrophy of the bowel is often seen above the seat of an obstruction, owing to the increased demand put upon the muscular fibres.

AMYLOID DEGENERATION.

Amyloid degeneration is not infrequent. It is most commonly met with in phthisis, when it is usually associated with tubercular ulceration of the gut. This causes very troublesome diarrhoea.

CIRCULATORY DISTURBANCES OF THE
INTESTINES.

PASSIVE CONGESTION.

Passive congestion is caused by general venous engorgement or by congestion of the portal system, as in cirrhosis of the liver. It may cause ascites, diarrhoea or hæmorrhoids.

HÆMORRHAGIC INFARCTION.

Hæmorrhagic infarction of the intestine is caused by obstruction of the superior mesenteric artery, by embolism or thrombosis. The intestine becomes more or less deeply

congested and may be of a blue-black colour. The tips of the valvulæ conniventes are most affected, and the congestion usually passes on to necrosis and is rapidly fatal. Acute peritonitis is commonly present. The condition is not easy to recognise during life. There is usually diarrhœa, colicky pain, vomiting (which may be fæcal) and abdominal distension.

Treatment.—*Passive congestion* must be treated on general lines. *Hæmorrhagic infarction* is very serious, although operative interference by resecting the affected loop of bowel has occasionally proved successful.

INFLAMMATIONS OF THE INTESTINES— ENTERITIS.

In general enteritis it is rare for the inflammation to affect the whole extent of the bowel, but it is usually so widely distributed that it practically comes to the same thing. Inflammations are common in special parts of the bowel, *e.g.*, in the upper and lower parts of the small intestine and in the colon, and special names have been introduced to indicate this local incidence, *e.g.*, duodenitis, jejunitis, ileitis, colitis, proctitis, and appendicitis. Few of these require special consideration. The duodenum is rarely, if ever, affected apart from the stomach, and it thus becomes part of the primary stomach affection. The jejunum is never affected alone. The ileum is usually affected in all common and special forms of general enteritis, and though the inflammation may not extend higher or lower than this part of the bowel, the clinical manifestations in no way differ from an inflammation of a wider distribution, and hence it is sufficiently fully considered under the more general term enteritis. The colon may be the main or sole

seat of the inflammation, and certain forms of colitis thus require a separate description. Inflammation of the rectum, on the other hand, though it may be almost the only part of the bowel affected, does not require separate consideration. Tenesmus and profuse mucous discharges are among the most characteristic of its clinical manifestations, and thickenings, polypoid outgrowths, strictures, perirectal inflammations (ischio-rectal abscess) and fistula its most important sequels. The appendix, on the other hand, is a frequent site of inflammation, and particular attention must be given to it. It is necessary, then, to consider separately general and special forms of enteritis, colitis and appendicitis.

GENERAL ENTERITIS.

DEFINITION—Inflammation of the small intestine or of the small and large intestine combined (entero-colitis).

Etiology.—Enteritis may, like many other diseases, be spoken of as primary and secondary. The former signifies that the exciting cause acts firstly and directly upon the mucous membrane of the bowel, producing an inflammation thereof to which all the subsequent local and general manifestations of disease are directly attributable; the latter that the disease starts elsewhere, as in the peritoneum, afterwards spreading to the bowel, or is of a general character, in which the intestinal lesion is merely a more or less subsidiary and perhaps a non-essential part. (1) Of all the causes of primary enteritis, errors in the ingesta play the chief part. Actual poisons, such as mineral acids, carbolic acid, compounds of arsenic, mercury, lead and silver, may be swallowed, and by their caustic or irritant properties set up enteritis, but far more commonly it is due to errors in the ordinary diet—the meals may be irregular, or the food improperly masticated or unsuitable. Imperfectly digested food may cause irri-

tation mechanically, but its chief action is through the formation of chemical substances of a toxic nature. Enteritis is very common in infants and young children, but rare in adults. In children, unripe fruit is a common cause, and it probably acts both as a mechanical and toxic irritant. The mixed diet so often given to very young children by ignorant mothers acts in the same way. Cow's milk is another and far more frequent cause, partly because it is an imperfect substitute for the mother's milk (it contains too much proteid and too little carbohydrate) but chiefly because it is liable to contamination and thus may contain poisonous substances, hence enteritis is far more prevalent in infants who are artificially fed than it is in those who are naturally fed. These poisonous substances are more likely to be developed in summer than in winter, and enteritis shows an increase from May to July, gradually sinking during autumn to the lower level of frequency which prevails during winter and spring. A very virulent form is the "cholera infantum," which is a severe entero-colitis. It occurs chiefly in hot weather, like the other forms of summer diarrhoea, of which it constitutes about three per cent. But alterations in the temperature may act in another way, for it has been frequently observed that enteritis may follow upon a bodily chill or a fall in the atmospheric temperature of from twenty to thirty degrees, probably acting through depression of the intestinal cells, congestion of its blood-vessels, and modification of the succus entericus. (2) Changes in the constitution of the intestinal secretions are held by many to cause intestinal catarrh. Knowledge of these changes is very imperfect at the present time. "Fatty diarrhoea" is attributed to deficiency in the pancreatic secretion, and "bilious diarrhoea" to an excess of bile. (3) Among other causes of primary enteritis, bacteria must be mentioned. Many varieties have been found in the diarrhoeic stools, particularly members of the colon and proteus groups, and it is

probable that not one specific kind but many different kinds are concerned in causing primary enteritis, and that they do so more by inducing changes in the food and intestinal contents, whereby injurious products are formed, than by direct irritation of the intestinal mucous membrane. Direct irritation is probably produced in some cases. The intestinal epithelium is constantly engaged in destroying the bacteria present normally in the intestine. In health, a balance is maintained between the resisting powers of the cells and the attacking forces of the bacteria. Should this balance be disturbed by anything which lowers the former, *e.g.*, a chill, or increases the latter, *e.g.*, the associations of newly-introduced bacteria, irritation and inflammation of the mucous membrane may result. (4) Animal parasites may cause irritation by their movements, by slight traumatic lesions, or by their secretions. (5) Inertia of the intestine, particularly of the colon and rectum, leading to lack of intestinal secretion and prolonged retention of the fæces, which become hard and dry, may cause irritation and inflammation.

The causes of secondary enteritis are many. They may be grouped in the following way:—(1) *Extension from neighbouring organs*, *e.g.*, the inflamed peritoneum, an intussusception, or a hernia, &c. (2) *Infectious diseases*.—The bacteria of typhoid fever, dysentery, cholera, tubercle and pyæmia frequently cause enteritis. In the case of the first two the enteritis proceeds rapidly to ulceration, which in turn tends to increase the inflammation. In tubercle it may show itself only in the form of numerous ulcers, which are, however, prone to cause in their turn a general enteritis. In pyæmia the enteritis is caused partly by the bacterial emboli and partly by their toxins. In cholera it is mainly due to the toxins. (3) *Wasting diseases*.—In cancer, in severe and prolonged anæmia, and in nephritis with uræmia, enteritis may develop and may prove fatal. It is believed to be caused, in part at any rate, by an attempt at

elimination by the intestine of the poisons produced in these diseases through abnormal metabolism or otherwise.

(4) *Circulatory disturbances*.—Cirrhosis of the liver and chronic heart or lung disease may cause enteritis through congestion of the portal system. This variety is largely chronic.

Morbid Anatomy.—The mucous membrane is swollen and of a greyish colour. There is rarely any hyperæmia except over the tips of the valvulæ conniventes. The intestinal secretion is increased and the mucous surface is coated with mucus, which usually contains only a few leucocytes, though exceptionally it contains many. In this *catarrhal form* there are usually few leucocytes in the interstitial tissue around the glands and around the blood-vessels, but in suppurative varieties they are very numerous, both here and in the submucosa, and may form into circumscribed abscesses or constitute a diffuse infiltration. This suppurative enteritis is called *phlegmonous*. When the solitary and agminated lymph glands are much swollen the enteritis is called *follicular*. This may be present along with a general catarrhal enteritis or without it. In other cases the exudate is not serous or mucous but fibrinous, forming a grey patchy covering, particularly over the tips of the valvulæ conniventes, under which the epithelium is necrosed in varying degrees, often leading to superficial ulcers from separation of these sloughs. This *fibrinous, croupous* or *diphtheritic* enteritis, for it is known by all three names, usually occurs secondarily to some infectious disease, *e.g.*, pneumonia. When ulcers are found, as they may be in the catarrhal, the suppurative, the follicular, or the fibrinous forms, the enteritis is spoken of as *ulcerative*, and the ulcers themselves are called by special names referring to their origin: thus we have catarrhal ulcers originating in hæmorrhages or in acute focal inflammations in catarrhal enteritis; follicular ulcers arising in

the solitary glands; typhoid and tubercular ulcers occurring in these diseases; stercoral ulcers in long-standing cases of constipation; malignant ulcers in cancer, &c.

Mucous colitis, a remarkable form of enteritis, called generally *mucous colitis*, affects mainly the large bowel. The names membranous enteritis, tubular diarrhoea and mucous colic are also given to it, but the first is the best, because the chief change is the production of an excess of mucus in the large intestine. This is very tenacious and adherent to the mucous membrane of the colon, which is not known to be otherwise altered. The mucus is passed in the stools as strings or a continuous membrane, which may be of a tubular shape, like a cast of the bowel. It is shown microscopically to be pure mucus, with or without an admixture of altered epithelial cells.

Ulcerative colitis, another peculiar form of enteritis, practically restricted to the large bowel, is known as ulcerative colitis. Both in its morbid anatomy and in its clinical course it is practically indistinguishable from true dysentery, to which it probably belongs.

Psilosis or *sprue* is a tropical and chronic form of enteritis. The intestine is thin, friable and more or less transparent, the stools are bulky, pale and frothy, with an acid reaction. It attacks persons, particularly Europeans, who have lived for a considerable time in a tropical climate. An aphthous stomatitis is generally present also, and a diminution in the size of the liver and gradual loss of strength seem to be fairly constant. It is often called also "White Flux," and is thought by many to be identical with the "Hill-diarrhoea of India."

Symptoms.—These vary considerably in the different varieties of the disease, but in the acute forms diarrhoea, vomiting and pain are the most frequent local complaints, while generally there is fever, collapse or convulsions. The diarrhoea may be very severe. In exceptional cases

it is absent. It commonly lasts for a week or so, though in the more chronic cases it may continue for prolonged periods. The stools vary greatly in number, colour and consistence. When they contain undigested particles of food the diarrhoea is called "lienteric." The stools may be very thin, but they are rarely serous, except in "cholera infantum," which forms about 2 to 3 per cent. of the acute summer diarrhoeas of children. Vomiting is generally absent, except in the severe forms of summer diarrhoea, particularly in cholera infantum, in which it may be very severe. Loss of appetite and thirst are generally present in all forms. Pain is often present, and is usually of a colicky character and accompanied by borborygmi. Fever is often slight or absent; on the other hand, it may be marked, the temperature rising quickly to 104° or 105°F . in children. In all cases the rectal temperature should be taken, as it may be two or three degrees higher than the axillary, particularly in cholera infantum. Collapse does not usually occur except in weakly children, but it may come on rapidly in very severe cases and cause death within twenty-four hours, particularly in cholera infantum. Convulsions may appear before the end in such cases, but they are uncommon otherwise. In the chronic forms, such as "mucous colitis," pain is frequent, but the main symptom is the passage of mucus, which may last for months or years without seeming to affect the patient's general health, except to increase nervousness, and the patients, mostly women and children, are often naturally of a nervous disposition. In sprue, the bulky, frothy white stools, somewhat like porridge, are the chief disorder, the stomatitis, diminished liver dulness and asthenia gradually following.

Treatment.—If there are irritant ingesta present in the bowel, an attempt should be made, by administering some purge, such as castor oil, to remove the irritant as promptly as possible. Where, as may be the case in

children, unripe fruit has been eaten, an emetic may prevent a part from passing beyond the stomach. In most cases, however, the first indication is to check the diarrhœa, which is often accompanied by great pain and also vomiting. Opium should be freely exhibited, and in severe cases is best given hypodermically. Bismuth, hydrocyanic acid and other sedatives may be freely administered, while ice given to suck and also applied to the abdomen affords great relief to the sufferer. Where there is no vomiting, salol or β -naphthol obviates the tendency to fermentation, and is often of great service in checking the diarrhœa. In addition to opium, lead acetate or zinc oxide may be used; sometimes one remedy succeeds better than another. It should not be forgotten that morphia suppositories ($\frac{1}{2}$ to 1 grain) often control the diarrhœa when all else has failed, and where the inflammation is limited to the lower part of the bowel washing out the intestine has proved efficacious. A number of different solutions may be used for this purpose, either nitrate of silver (which is extremely painful) or warm boric lotion.* Starch and laudanum administered as a small injection (1 to $2\frac{1}{2}$ ounces in quantity) is very soothing and may precede and follow the more painful injections. For *ulcerative colitis* there is no better method of treatment than irrigation, although it is difficult to induce the patient to submit to the inevitable pain. Where there is *lienteric diarrhœa* 2 to 4 minims of liquor arsenicalis should be given with food two or three times a day. In *mucous colitis* there is no remedy so helpful as the administration of sulphur, which may be given as the compound liquorice powder (1 to 2 drachms) or equal parts of sulphur and cream of tartar ($\frac{1}{2}$ to 1 drachm of each). The patient suffering from this latter affection is often hysterical, and this feature of the case may demand the attention of the physician. In *sprue*, rest and milk diet are usually prescribed.

* See treatment of "Dysentery," page 210.

In all cases of enteritis, no matter what type is under treatment, diet is all-important, and the elimination of everything which irritates, and the gradual introduction of a dietary not merely sufficient to support life, but which will tend to sooth the inflamed bowel, should receive the physician's careful attention. Yet another matter is the clothing; no sufferer from enteritis should be exposed to any chance of chill, and to this end the wearing of a cholera belt, woollen underclothing in cold weather, warm socks or stockings, and water-tight boots are imperative.

APPENDICITIS.

Inflammations in the region of the cæcum, formerly known as *perityphlitis*, a name still preferred by some, are now known to be in nearly all cases due to disease of the vermiform appendix.

Etiology.—(1) *Sex*.—Appendicitis is more common in males than in females. (2) *Age*.—While it may occur at any age, the great majority of cases occur between ten and thirty years of age. This is partly accounted for by the richness of the appendical lymphoid tissue in youth and its gradual atrophy after thirty. (3) *Vulnerability*.—The lymphoid tissue is the most vulnerable part of a very vulnerable organ. The anatomical relations and history of the vermiform appendix (it is a gradually involuting or disappearing organ) and the small size of its lumen partly account for this vulnerability. (4) *Vascular disturbances*.—The appendix is liable to become kinked and thus congested. The main artery, which runs along the free margin of its mesentery, may become obstructed (though this is believed to rarely occur) and the vitality of the appendix may be consequently diminished. (5) *Fæcal concretions and foreign bodies*.—The latter are rare. The former are

common, particularly in habitual constipation, and consist of inspissated fæces and mucus, occasionally with leucocytes or epithelial cells. They are liable to become impregnated with lime salts. They may be expelled from the appendix by vigorous peristalsis, with or without injury to the mucous membrane. Some cases of "appendicular colic" may possibly have this explanation. The injury to the mucous coat may not be properly repaired, and in that case it may lead to the formation of fresh concretions, which bring about further injury to the mucous coat, so that it falls an easy prey to micro-organisms. (6) *Germes are probably in all cases the real producers of appendicitis.*—The ground is prepared for them in one or other of the ways mentioned above, and their own virulence is at the same time in all probability gradually increased by their adaptations to the altered conditions of the mucous coat and its secretions. The bacillus coli is most frequently (94 per cent.) present, the pyogenic cocci, viz., the staphylococci and streptococci, coming next, while the diplococcus lanceolatus, the typhoid and the tubercle bacillus are also found. Any of these germs may act alone or in combination. They are mostly natural inhabitants of the intestinal canal, and hence the conditions which lead to their heightened virulence and those which produce diminished vitality of the tissues of the appendix are of chief importance. Surgeons have found that the appendix may become greatly swollen and oedematous in its efforts to get rid of any obstruction to its lumen, and this change, along with the mechanical injury to its wall, may be favouring conditions of much influence.

Morbid Anatomy.—The morbid process may run an acute or chronic course. The acute inflammation may be classified as follows:—(1) Catarrhal, (2) Suppurative, (3) Ulcerative, (4) Gangrenous. The catarrhal form is mainly limited to the mucous coat and shows a desqua-

mation of the lining epithelium at one or more places, a dilatation of the blood-vessels, and a swollen appearance of the lymphoid tissue, due at first to œdema and later also in many cases to cellular proliferation. There may be some œdema of the submucous and other coats, but the peritoneal coat and mesentery look unchanged. The lumen is compressed. In the suppurative form there is, in addition, a diffuse leucocyte invasion of all the coats, particularly of the submucous, and there is generally some lymph externally, while the lumen may contain pus or muco-pus. The pus naturally makes its way towards the lumen. The ulcerative form may follow upon the last or, as is more frequent, arise independently. The change is more localised. A focus of necrosis occurs usually at one place (probably the site of a concretion), but sometimes at more than one. An ulcer is formed which implicates the wall of the appendix over a variable area and to a variable depth, and may even penetrate it. The gangrenous form usually implicates a large part of the organ, converting it into a black slough. The affection of the appendix is of little importance in itself, but of great moment when the inflammation spreads to the surrounding structures. In some cases, owing to the position of the appendix, *e.g.*, retro-cæcal, the resulting inflammation affects extra-peritoneal tissues and an abscess situated outside the peritoneum is produced, but in most cases the extension implicates the peritoneum, usually, however, only locally in the neighbourhood of the appendix, due to fibrinous adhesions shutting off the general peritoneal cavity prior to the formation of pus. A peri- or para-appendical peritoneal abscess is thus formed. This fortunately happens in most cases. In some of the suppurative, some of the ulcerative, and in more of the gangrenous forms the process is unfortunately too rapid to allow of this, and a fatal general peritonitis ensues. If the local abscess is not artificially evacuated it will enlarge and may empty itself into the cæcum or other part of

the bowel, or perforate through the skin or into the peritoneum, or extend by means of suppurating tracts beneath the cæcum or anywhere else in the abdomen, the pelvis, the rectum, the gluteal region, &c., and even to the diaphragm, and there form a subphrenic abscess or even extend through the diaphragm into the pleura or lung. Suppuration in the omentum or portal vein, both outside and within the liver (suppurative pylephlebitis), may ensue. Chronic appendicitis leads in time to obliteration of the lumen of the appendix and the conversion of the organ into a fibrous-like cord. Whether it follows upon an acute or sub-acute attack or is chronic from the start, the changes soon become much the same. The lining epithelium is lost and the mucous and sub-mucous coats are converted into granulation tissue, which is gradually replaced by permanent fibrous tissue. These chronic cases are usually steadily *progressive*, but in some cases the process subsides, and after a period of quiescence starts again, giving the relapsing or recurrent form of the disease. The extent of the appendix affected by chronic inflammation varies. In some it implicates the whole organ, in others a part only, and when it is confined to the proximal end the rest of the organ may become greatly distended with its own secretions and form a cyst-like structure.

Symptoms.—They resemble those of peritonitis, and it is possible that the changes in the appendix itself gives rise to no symptoms, and that these arise only when the inflammation spreads to the surrounding peritoneum or extra-peritoneal tissues. If this be so, most cases of catarrhal appendicitis, however acute, must come and go unnoticed. *Pain* is alike one of the earliest and the most striking symptoms of appendicitis. It is mostly due to involvement of the peritoneum; but its explanation is uncertain, for it is not constantly present in such cases, and

it is, moreover, sometimes present and of varying intensity and duration when the appendix is diseased without any change in the peritoneum. It may in these cases be due to the inflammatory œdema of the appendical walls pressing upon the nerve endings. The typical pain of appendicitis is severe and paroxysmal. It usually comes on without warning, either when walking or at night-time (frequent), particularly after a full meal. At first it may be general in the abdomen or be chiefly about the umbilicus, but it soon becomes localised to the cæcal region, though it radiates into the loins, pelvis and thighs, and even to the left iliac fossa, where it may be very severe. The cæcal region is *tender* on pressure, the *maximum tenderness* often being at M'Burney's point. A palpable and visible swelling in this region soon follows, though it may not be detected for several days on account of the tenderness. *Fever* is almost invariably present, at any rate at the beginning. The rise of temperature may be moderate or, particularly in children, great, but it follows rapidly upon the pain and runs an irregular course, usually for eight or ten days, when it generally subsides. The *pulse* is quickened in proportion to the fever, but may remain quick even after the temperature falls. *Nausea* and *vomiting* are frequent, though sometimes absent altogether. They usually subside by the second day in favourable cases, but continue in the acute perforative cases. Constipation is the rule. There is sometimes great irritability of the bladder. The patient lies on the back with the right leg, or both legs, flexed. The acute symptoms gradually subside spontaneously in the great majority of cases. Improvement begins in many as early as the third or fourth day, in others as late as the eighth or ninth, but the acute symptoms have generally subsided within a week to a fortnight, and convalescence is established within the month. A certain proportion, not a large one, of all the cases proceed to suppuration, with the formation of an

abscess as early as the third or fourth day, or as late as the third week, though usually about the fifth or sixth day. There is no individual sign diagnostic of suppuration except actual fluctuation in the tumor, but certain signs are suggestive thereof—(1) an increase, particularly if rapid, in the size of the tumor; (2) an aggravation of the general symptoms; (3) an increase in the temperature; (4) *a marked increase in the rapidity of the pulse*, say 120 or over, particularly if the temperature does not rise proportionally; (5) a well-marked leucocytosis. A still smaller proportion of cases are *ultra-acute* and have been termed *fulminant*. They are actually rare when compared with the suppurative ones, and still more rare when compared with the non-suppurative ones, which constitute the vast majority. The symptoms begin suddenly without any warning in apparently perfect health and become at once severe. They are due to the onset of a general peritonitis from a perforated or gangrenous appendix. There may be little fever or pain, but vomiting is generally very marked, the abdomen becomes distended, the pulse rapid, and collapse may end in death in thirty-six to forty-eight hours from the first onset of the symptoms. Most of the *fulminant* cases run this course, but a few begin moderately and remain so for a time, showing that the peritonitis is local at first, but after one or more days severe symptoms ensue, showing that the peritonitis has become general.

Diagnosis.—While difficult at first, it becomes in most cases easy as the symptoms become marked. Pain of sudden onset in the umbilical or cæcal region, with fever and localised tenderness, are the most reliable symptoms. When vomiting, constipation and a distinct tumor are also present there is little room for doubt. Fulminant cases are difficult to distinguish from a perforated gastric or duodenal or typhoid ulcer, from intestinal obstruction,

and from acute hæmorrhagic pancreatitis. Other forms may closely simulate pelvic peritonitis (particularly inflammations of ovary or tubes), perinephric abscess, renal or biliary colic, or even hip-joint disease with suppuration. A careful analysis of the symptoms and a comparison with those characteristic of these diseases will generally enable a diagnosis to be made. In a small number of cases the symptoms may be misleading or almost absent, and even a large collection of pus may be unexpectedly found. *Pain* about M'Burney's point and *tenderness* in the cæcal region coming on suddenly in a perfectly healthy child are particularly suspicious of appendicitis. The common practice of giving an aperient to children should be avoided in such cases.

Prognosis.—This must always be guarded, for, though the great majority of cases run a favourable course and recover without operation, and a considerable number recover with it, and only a comparative few die, the mildest case may quickly become dangerous. It is estimated that 5 per cent. represents the rate of mortality of all cases, that 80 per cent. of the fulminant cases and 30 per cent. of the abscess cases die, but these results are likely to improve under more timely surgical influence.

Treatment.—There are obviously two lines of treatment—*surgical* and *medical*. The *medical* treatment consists in the application of ice to the abdomen, the use of rectal enemata, and the administration by the mouth, should there be no vomiting, of some sedative such as opium or belladonna. Opium may be injected hypodermically, but in whatever way it is used it must be given with the greatest caution, because it is apt to mask symptoms, and therefore many physicians prefer not to give it at all. The *surgical* treatment must be sought for in surgical text-books. It is true that operative interference is not

necessary in every case, and many patients only have one or two attacks of appendicitic pain in all. Do not advise immediate operation if twelve hours have elapsed after the beginning of an attack and the symptoms are not urgent. Should vomiting continue, or should the case be a fulminating one, or perforation be imminent, operate at once. Unfortunately we cannot always foretell if any particular case is going to be of the fulminating type, and therefore it is difficult to be dogmatic as to the propriety of operating in any particular case. It seems probable that constipation and intestinal fermentation are responsible for many of the milder recurrent attacks of appendicitis, and therefore intestinal antiseptics and the systematic administration of purgatives may sometimes prove sufficient to obviate further attacks.

SPECIAL INFLAMMATIONS OF THE INTESTINE.

Typhoid has already been considered, but tubercle, syphilis and actinomycosis require notice. Tuberculosis of the intestine is very frequent. It is rare as a primary affection, except in children, who may be infected through milk containing either the human or bovine bacillus. It is frequent as a secondary process to tubercle elsewhere, particularly the lungs, rarely the peritoneum. The most marked changes are found, as in typhoid, near the ileo-cæcal valve, but they may extend from the duodenum to the anus. The Peyer's patches and solitary glands, which the disease specially affects, show minute nodules beneath the mucous membrane, at first grey and translucent, soon enlarging and becoming white or yellow (caseous). The caseous material becomes softened and discharged, leaving an ulcer with thickened margins. Similar foci

form around the ulcer, and, breaking down, in turn extend the size of the original ulcer. This extension is usually transversely to the long axis of the intestine because the lymphatics and blood-vessels tend to run round the bowel. The lymphatic extension is well seen on the peritoneal surface of the floor of the ulcer in the form of white or yellow lines (the injected lymphatics) radiating transversely outwards from the centre. The floor of the ulcer is rough and nodular and generally reaches into the muscular, and often also to the peritoneal, coat, but rarely actually perforates into the peritoneum. This latter contingency would happen oftener than it does were it not that adhesions of the floor to the omentum, mesentery or neighbouring coils of bowel generally prevent it. Old ulcers may partially cicatrize and cause constriction of the bowel at one or more points. Fistula in ano is often caused by the perforation of a tuberculous ulcer of the

TABLE SHOWING THE CHARACTERISTICS OF FOUR FORMS OF INTESTINAL ULCERS.

TYPHOID ULCER.	TUBERCULAR ULCER.	DUODENAL ULCER.	DYSENTERIC ULCER.
<p>The long diameter is parallel to the long axis of the intestine, and the edges are thin, ragged, and undermined. These ulcers do not lead to stricture of the intestines, and, as a rule, heal readily. They are seldom accompanied with local or general peritonitis, have a smooth floor and often perforate the intestine. The ulcers are limited to Peyer's patches.</p>	<p>The long diameter is transverse to the long axis of the intestine. The edges are thick, undulated, indurated, not undermined. The ulcer often precedes stricture, and seldom heals, and is, as a rule, accompanied with local or general peritonitis. The floor of the ulcer is rough and nodular, and perforation is rare. The ulcers are not limited to Peyer's patches.</p>	<p>The orifice of the ulcer is circular, from three to six or more lines in diameter, with a sharp peritoneal edge as if a round piece of the intestine had been punched out. The edges of the ulcer seem bevelled off from within outwards; may perforate intestine, giving rise to peritonitis.</p>	<p>The ulcers are round, oval, or irregular, with infiltrated, undermined edges. The opening of the ulcer is smaller than the cavity. The floor may be formed by the submucous, muscular or serous coat of the intestines. The ulcers are found in the different portions of the large intestines.</p>

Symptoms.—They are not very distinct. Diarrhoea, colicky pains and fever seem to be most commonly observed. It is the primary cases in children which are most difficult to diagnose, and prolonged observation is generally required before it can be done. Extensive ulceration may exist and severe or even fatal hæmorrhage may occur without any warning, and it is probably to a consequent general enteritis that the symptoms are mostly due. The signs may be those of appendicitis when the main affection is in the cæcal region. Constipation may be present, and when diarrhoea is marked it may in part be due to a co-existent waxy degeneration of the intestinal wall.

Syphilis of the intestine most frequently affects the rectum near the anus, either in the form of papules, condylomata, or a gumma. The latter leads to ulceration and cicatrization, with consequent stricture. The symptoms are those of ulceration or stricture, according to the stage. Actinomycosis of the intestine is rare. It causes the formation of white patches beneath the mucous membrane, which ultimately break down and form ulcers. It is liable to extend to the peritoneum and also to the liver, causing abscesses therein, also to the ovary, and it is probable that all the cases of actinomycosis of the ovary are secondary to the bowel. When fistulous openings occur externally a diagnosis may be made, but not otherwise, except after operation.

Treatment.—The treatment depends on and varies with the nature of each case. Not a few of the conditions grouped together here only admit of palliative measures; where operation is necessary the surgeon's help should be sought without delay.

TUMORS OF THE INTESTINE.

Mucous polypi are not uncommon in young children, but cancers are the most important tumors of the intestine. They rarely occur in the small intestine, except in the duodenum at the biliary orifice. In the large intestine they occur chiefly at the flexures, viz., the cæcal, hepatic, splenic, sigmoid and rectal. They form a sharply-defined tumor, rarely, as in colloid cancers of the rectum, a diffuse infiltration of the wall. Ulceration of the surface parts of the growth takes place, and as the tumor extends round the bowel stricture follows, which gradually gets greater and greater until it results in obstruction of the bowel. The symptoms are chiefly those of chronic intestinal obstruction. Considerations of space do not permit description of a few of the rarer intestinal diseases, but it is advisable to add a short account of such prominent intestinal conditions as diarrhœa, constipation and colic, although they are not in themselves diseases.

DIARRHŒA.

This signifies an abnormal frequency and looseness in the action of the bowels.

Etiology.—The causes are generally grouped into the two heads—(1) an increased intestinal peristalsis, (2) an unusual fluidity of the intestinal contents. Most cases are due to some combination of these causes. The factors which bring about an increased peristalsis may be—(a) stimulation by the intestinal contents, (b) lesions in the intestinal walls, (c) irritants circulating in the blood, and (d) perverted innervation. Those which bring about excessive fluidity of the intestinal contents act chiefly by

increasing the intestinal secretion, though they may also act by diminishing the natural absorption of water from the bowel (which mainly takes place in the colon). The hypersecretion may be caused by saline purgatives, by passive congestion of the bowel, as in hepatic cirrhosis or cardiac disease, by toxic irritants circulating in the blood, as in the critical evacuation of pneumonia, or produced *in situ* in the intestinal canal, as in cholera, and, lastly, by perverted innervation. Most cases of diarrhœa will find explanation in one or other of these causes, or in some combination of them.

Symptoms.—The condition may be acute or chronic and of all degrees of severity. The number of evacuations may be few or many, and their colour and consistence also vary greatly. Pain may be present or absent. The general health suffers when the diarrhœa is acute and much fluid is lost and when it is prolonged over long periods. In very acute cases collapse may rapidly supervene, as in Asiatic cholera and cholera infantum.

Treatment.—Many are the causes of diarrhœa, and the treatment for most of these will be found under such headings as typhoid fever, cholera Asiatica, dysentery, enteritis, &c. In not a few instances a dose of castor oil or other purgative clears out any intestinal irritant, and is an excellent preliminary measure. To arrest an acute attack of diarrhœa no remedy is so efficacious as opium, and the best preparations administered by the mouth are the pulvis cretæ aromaticus cum opio in 10 to 30 grain doses, the pulvis ipecacuanhæ compositus in 5 to 10 grain doses, and bismuth subnitrate in large doses, which may be combined with one of the above or given separately. The lead and opium pill of the British pharmacopœia is often useful, and lead acetate, copper sulphate and other powerful astringents may also be given. In other cases

treatment by the rectum is preferable, and opium suppositories and laudanum and starch enemata act promptly and may be used where remedies cannot be retained by the stomach. Reference has been made under dysentery to the value of large injections of silver nitrate solutions, and certainly where the large intestine is the probable site of any inflammation, lavage with warm boric lotion is often of great value. In the diarrhœa of children this irrigation method has been found very successful in not a few cases.

Where there is much fermentation, salol and charcoal may be given by the mouth, and in children the well-known pulvis infantum, consisting of hydrargyrum cum creta (1 to 2 grains), pulvis ipecacuanhæ compositus ($\frac{1}{2}$ to 1 grain), pulvis rhei (1 grain), and pulvis cinnamomi compositus (1 to 3 grains) is prompt in action, first in removing any irritant in the bowel and then in arresting the diarrhœa. For the green stools of very young children there is probably no remedy so successful as dilute hydrochloric acid in doses of 1 to 5 minims.

The diet is an important part of the general treatment of every patient suffering from diarrhœa. An attempt should be made to increase the intervals between meals, to afford some rest to the bowel, and the food given should contain as little waste matter as possible. Probably milk and lime water or peptonised milk are amongst the most suitable methods of feeding, and the patient's strength may be supported by the administration of iced whisky or other form of alcohol. A good deal of attention has lately been paid to the various kinds of organisms in the stools of infants and young children, and if it is the case—and there seems to be almost conclusive proof thereof—that certain forms of epidemic diarrhœa are directly the result of the infection of the bowel by specific organisms, an attempt should be made by means of suitable anti-septic treatment to effect their removal.

CONSTIPATION.

This is an insufficient emptying of the bowels. In the majority of people an efficient evacuation occurs once a day, but this is largely a matter of habit, and it is important to ascertain the individual habit in each case. Every three or four days is the habit of some, and longer intervals, even to weeks or more, is the habit of others. The frequency does not matter provided the bowels are sufficiently emptied each time.

Etiology.—The causes are generally classified somewhat artificially, though usefully, into *general* and *local*. The former include sedentary habits, inattention to the calls of Nature, anæmias, and all enfeebling diseases, certain nervous diseases, such as meningitis, tabes dorsalis, neurasthenia, hysteria, &c., as well as other diseases, such as the acute fevers, chronic affections of the heart, liver, &c., but it is probable that they all act through the neuromuscular apparatus of the intestinal canal and thus bring about a diminution of intestinal peristalsis. In this sense all the causes of constipation are local and act either by weakening the muscular power of the intestinal or abdominal walls or by diminishing the *succus entericus*. Diminished peristaltic power may be caused by degenerations, congestions, catarrhs, tumors, strictures and other lesions of the bowel, or by inflammation of the peritoneum or by drugs, such as opium, or prolonged use of aperients. Atony of the intestine, particularly of the colon, which may result from any of these lesions, is a very frequent cause of constipation. Weakness of the abdominal muscles is met with in great obesity, after many pregnancies, after great distensions of the abdomen by fluid or tumors. Diminished intestinal secretions are the result of chronic catarrh and atrophy, of astringent drugs, &c.

Symptoms.—They are extremely variable, and this is only natural when it is remembered that constipation itself is only a symptom result of many different diseases. A marked degree of constipation may exist without appearing in any way to affect the comfort or health. In other cases there may be gastro-intestinal symptoms, such as a furred tongue, loss of appetite, a sense of distension or fulness or pain in the abdomen or pelvis, and large masses of fæces may be felt in the colon, particularly in the cæcum and hepatic and splenic flexures. There are often also cerebral symptoms, such as headache, lassitude and mental depression.

Treatment.—Constipation may be due to numerous causes, and it is requisite to keep these in mind in prescribing appropriate treatment. The constipation forming a part of intestinal obstruction is referred to under that heading, and nothing need be said here as to procedures for its relief. Where a patient lives a sedentary life, more exercise and, if circumstances permit, riding, gymnastics and other healthful pursuits do much to relieve the constipation. The first matter demanding special attention is a very important one, namely the neglected habit of going to stool *daily* and at some *regular hour*. If this habit has been given up, order the patient to resume it at once, even although there is no inclination at the time. In men a pipe after breakfast often aids an evacuation of the bowels. Where a patient is in feeble health, or unable to take sufficient exercise, the abdominal wall may be massaged. A valuable method of carrying out this massage is to roll a hollow wooden ball, which may be filled with a greater or less amount of shot, over the abdominal wall. When either massage with the hand or the ball is being carried out, it is well to remember the line of the large intestine and the direction of normal peristalsis. In persons whose abdominal wall is flabby, or who are excessively stout, a broad supporting bandage is

found to be beneficial. Discourage the too frequent use of enemata, and where it is requisite to assist Nature, prefer drugs which encourage normal healthy action. Much may also be accomplished by means of diet, ordering for the patient foods which leave a considerable portion of indigestible waste matter. Brown bread, porridge made of not too finely ground meal, certain fruits, and especially figs and prunes, vegetables such as cabbage, salad, &c., are all very efficacious. The amount of fluid in the intestine may be increased by drinking a tumblerful of warm or cold water before breakfast every morning. Not a few cases of constipation can be relieved by careful attention to these measures without having recourse to drugs at all.

The best drug from the point of view of increasing natural peristalsis is cascara sagrada, because it acts as a tonic to the lower bowel, and the daily dose may be gradually diminished until the patient can do without it altogether. Strychnin and belladonna form a useful combination for a pill. Among less satisfactory purgatives are salts of different kinds. Magnesium sulphate in doses of 2 to 8 drachms taken in a tumblerful of warm water should produce a free aperient action. Carlsbad salts, Apenta and many other purgative mineral waters or the salts obtained from them may be prescribed, and a course of such treatment is often of use, although, if salts are persisted in, a habit is formed, and the bowel refuses to act without the usual assistance. Amongst other remedies which might be mentioned are castor oil, aloes, and jalap. Castor oil may be taken in capsules, or if suspended in a wine-glass between a small quantity of cinnamon water and a little brandy it can be swallowed almost without the patient perceiving the taste. A little lemon juice or a small piece of dry bread are other well known methods of concealing the nauseous taste of this drug. Where hard scybalous masses are present, 10 or more ounces of warm olive oil should be injected and retained

for half-an-hour or longer, the patient remaining in the recumbent posture. This must be followed by a large soap and water enema, and these injections may have to be repeated several times before relief is obtained. A small injection of glycerine (1 drachm) is a favourite method of treatment in mild cases of constipation. Glycerine or soap suppositories may also be prescribed, and the latter form a useful addition to the purgatives used in the nursery.

The method of administering an enema requires a few words of description:—the patient should be placed on his left side with the hips raised on a pillow, and the fluid should be slowly injected, with occasional halts when any pain is produced. He should be directed to retain the enema for a few minutes if possible.

INTESTINAL COLIC—ENTERALGIA—BELLY-ACHE—THE GRIPES.

Etiology.—Indigestible food or iced drinks may be followed by abdominal pain. Lesions of the bowel wall, such as enteritis, new growths, displacements leading to intestinal obstruction, &c., may be associated with pain. Gout and lead poisoning are often similarly associated with abdominal pain. It is thought that this is sometimes due to local spasm of the bowel, mostly the colon, hence the term “colic.” Similar paroxysmal attacks of griping pain occurring in the cæcal region have been described as “appendicular colic,” under the belief that they originate in the appendix, and there seems reason to believe that in many of these cases at any rate there may be an acute, though slight, inflammation of its mucosa. It is possible that colic may be caused by an abnormal stimu-

lation of the intestinal nerves without any inflammation or other detectable lesion of the bowel wall.

Symptoms.—The pain is usually severe and paroxysmal, lasting only for a few minutes, but recurring once or oftener at irregular intervals. It is most frequently situated near the umbilicus and radiates in various directions. It is described as “griping,” twisting, tearing, boring, &c., and causes the patient to double up his body. Pressure over the abdomen generally gives relief. There may be flatulence, the passage of which, or of a subsequent stool, also affords relief, often unbroken by a subsequent attack. Beads of cold perspiration may break out on the forehead, the pulse tends to become small and frequent, and collapse or convulsions, particularly in children, may ensue.

Treatment.—Colic is a sign of many serious ailments in addition to being a common result of the presence of an intestinal irritant. In the less severe cases the first duty is to remove the irritant, and this is commonly done by the administration of a full dose of castor oil, along with 20 to 30 minims of laudanum: should this not prove promptly beneficial an enema may be given. Fomentations or linseed poultices applied over the abdomen afford great relief, and intestinal antiseptics such as salol, charcoal and β -naphthol may prove of benefit.

TABLE giving the Diagnosis in Renal Colic, Hepatic Colic, and Intestinal Colic.

RENAL COLIC.

Aching burning pain in one or other or both lumbar regions or side of abdomen, shooting down to the testis and labium and inner side of thigh. Frequent micturition, soreness and pain during the act, especially at the end of the urethra. Urine may be passed clear, but deposits a sediment of crystals and epithelial scales. In some cases the urine may contain blood. Nausea and vomiting. The pain may end suddenly, due to the calculus being arrested in the ureter or entering the bladder.

History of previous attacks.

HEPATIC COLIC.

Severe pain, coming on suddenly, often after a meal, with remissions and exacerbations lasting a few hours or a day or two. Faintness, nausea, and vomiting. The pain may be slight or so severe that the patient is bathed in a cold sweat. The pain is referred to the pit of the stomach or to the umbilicus, thence extending to the back and shoulders. The vomit often contains bile, and the stools are clay coloured. The urine may be tinged with bile, and jaundice may also occur. Due to the escape of the gall-stone into the bowel or its slipping back from the duct into the gall bladder; the severe pain may suddenly cease, leaving only a sensation of soreness or tenderness to the touch. The pulse may be weak and feeble.

History of previous attacks.

INTESTINAL COLIC.

Severe twisting, dragging or wringing pain referred to the umbilicus or upper part of the belly, in most cases relieved by pressure. The pain is often paroxysmal, during the paroxysm the patient leaning with the whole weight of his body across the back of a chair or other support. Nausea and vomiting are often present, generally of undigested food, giving relief when the latter occurs. Flatulence is, as a rule, present, giving rise to "borborygmi" or rumbling noises in the abdomen. There is little or no constitutional disturbance.

History of previous attacks.

VII.—DISEASES OF THE LIVER.

DEGENERATIONS.

The usual degenerations, *e.g.*, cloudy swelling, fatty degeneration and infiltration and waxy disease occur in the liver. Both fatty and waxy change may give rise to such great increase in size of the liver that it becomes a prominent organ in palpation of the abdomen, but they cannot be said to be clinically important apart from the general diseases with which they are associated. Their causes and morbid changes have been sufficiently considered in the early part of this book.

ALTERED CONDITIONS OF THE CIRCULATION.

HYPERÆMIA.

Hyperæmia may be active or passive. The former occurs periodically during digestion. This is a physiological condition, but though pathological hyperæmias probably also occur, their causes and results are too little known to merit discussion.

PASSIVE HYPERÆMIA.

Causes.—It occurs in all cases of obstructed return of blood to the heart or through its various chambers, hence it is seen in many cardiac and chronic lung diseases.

Morbid Change.—The blood accumulates in the liver, distending the hepatic, sublobular and central veins and hepatic capillaries in turn. This dilatation is greatest in the centre of the lobule and leads to gradual atrophy of

the liver cells and some thickening and pigmentation of the capillary walls. The liver is enlarged in the earlier stages and may remain so for a considerable time. Later it becomes smaller and firmer.

Symptoms.—There may be no symptoms apart from those of the primary disease, but gastro-intestinal catarrhs are apt to occur. Hæmatemesis may follow, and in the later stages also ascites; but it is doubtful if the ascites is due alone to the congestion of the liver, or even to the general congestion of the portal system. Indeed, it is very probable that one or more factors connected with the general disease are fully as much or more to blame.

Treatment.—Indications for treatment are chiefly directed to the use in the first place of purgatives and diuretics, and in the second to the administration of cardiac tonics such as digitalis. Sometimes the tapping of pleuritic, ascitic or other dropsical effusion may also be necessary.

THROMBOSIS AND EMBOLISM.

The liver is supplied with blood by both the hepatic artery and the portal vein, hence the circulation, though the rate of flow of blood in its capillaries is slow, is very free, and simple infarcts like those in the spleen and kidney do not occur. It is true that an infarct of the liver has occasionally been seen in embolism of the hepatic artery when there has been pronounced cardiac weakness, but this and other affections of the hepatic artery, with the exception of liver abscess resulting from a septic embolus in cases of malignant endocarditis, however interesting they may be pathologically, are not of clinical importance. It

is different in the case of the portal vein. Simple thrombosis is indeed rare, occurring only in certain cases of hepatic cirrhosis, at any rate to any extent, but suppurative thrombosis and emboli are of comparative frequency. When the emboli are small they get caught in some of the hepatic capillaries, generally those near the surface of the liver, where they give rise to necrotic foci or abscesses. When the disease is more severe and prolonged they affect larger branches and cause a suppurative thrombo-phlebitis or pylephlebitis and abscesses in the surrounding liver tissue. They arise in affections (*e.g.*, ulcerative and suppurative inflammations) of the intestine, appendix, stomach, spleen, pancreas and pelvic organs, and will be further considered under "Abscesses of the Liver." The umbilical vein of new-born infants is another vascular channel by which infection may reach the liver, causing suppurative phlebitis and poisoning. This is believed to be one of the causes of the severe form of *icterus neonatorum*.

INFLAMMATIONS OF THE LIVER—ACUTE AND CHRONIC HEPATITIS.

ACUTE HEPATITIS.

It is difficult to draw lines between degeneration, acute congestion and acute simple inflammation of the liver, and the same causes may at times produce the one or the other. Bacteria are constantly reaching the liver from the intestine, and are, in the healthy state, as constantly being destroyed therein, but this bactericidal action may be interfered with by various causes, such as over-indulgence in rich foods and in strong alcoholic drinks, residence in hot climates, &c. In other cases the bacteria are brought to the liver in number and virulence too great for its powers, as in

such general diseases as pyæmia, malaria, typhoid fever, or in such local diseases as appendicitis, dysentery or other intestinal ulcerations. The changes in the liver will depend upon the severity of the infection. In mild cases epithelial degeneration, dilatation of the branches of the portal vein, hepatic artery and hepatic capillaries and slight leucocytic interstitial infiltration occur, while severe cases will terminate in suppuration.

Symptoms.—The symptoms of the milder acute inflammations and acute congestions are thus alike and of an ill-defined character. There may be some dull pain and heaviness in the region of the liver, some fever, and some enlargement of the liver: symptoms, in short, often summed up as indicating “biliousness.”

Treatment.—The patient should be kept so far as possible to a very limited diet such as milk, and the remedies administered should include purgatives, diuretics, and, where necessary, diaphoretics.

SUPPURATIVE HEPATITIS.

Suppurative hepatitis or abscess in the liver, on the other hand, is so common and definite that it merits a somewhat full description.

Etiology.—It may result from the entrance of suppurative bacteria or the amoeba coli in sufficient numbers into the liver by any of the possible paths. Some of these have already been mentioned, *e.g.*, the hepatic artery in malignant endocarditis or in pyæmia, the portal vein in intestinal lesions, particularly dysentery and appendicitis, the umbilical vein of new-born infants, and there remains still to be mentioned the bile ducts (where a suppurative angiocholitis results in abscess) and parasites (where suppuration

occurs in an echinococcal cyst). In a few rare cases the infection has reached the liver backwards, as it were, from the heart through the inferior vena cava and hepatic veins.

Morbid Anatomy.—The abscesses may be single or multiple. The *tropical abscess* is usually single (though there may be several), of variable size, often large and deeply seated in the liver, tending to make its way through the capsule and diaphragm into the pleura or lung, though it may rupture through the capsule into the peritoneum, colon or other neighbouring structure, or even through the external skin. Its purulent contents are usually stained of a reddish or chocolate colour, so that they have been likened to mashed strawberries and cream or to anchovy sauce. The contents in many cases contain the amoeba coli, probably the causal germ, with or without one or more of the ordinary acute pyogenic germs, but in other cases the cause is obscure. Alcoholic excess and climatic conditions are considered by many to have an important influence, and some cases may be the local manifestation of a general infection. Other forms of abscess, *e.g.*, systemic or suppurative pylephlebitis or cholangitis, are usually multiple and easily traced to the branches of the hepatic artery, the portal vein, the bile ducts or to echinococcal cysts. In these cases the contents are more frequently like ordinary pus, stained, it may be, by bile, or foetid or thick, but still having the usual appearances of pus. The organisms present vary greatly, the ordinary pyogenic germs in pyæmic cases, the bacillus coli in biliary cases, the typhoid bacillus, the actinomyces, &c.

Symptoms.—I. OF THE LARGE SOLITARY ABSCESS.—Sweating is often profuse, particularly when the patient is asleep. Fever is the most important symptom. It is markedly irregular, often rising to 103° or more, and its intermittent character is apt to suggest malaria. Pain is

another but very variable symptom. It may be absent or present, and when present it is often referred to the right shoulder rather than to the hepatic region. It may be brought out by deep pressure or by making the patient lie on the left side, when it is usually a heavy sensation rather than an acute pain. In chronic cases all symptoms may be absent. Enlargement of the liver, particularly in an upward direction, is a sign of great importance. Fluctuation may be detected in large superficially situated abscesses. Marked jaundice is rare, but the skin has usually a pale, muddy, slightly-jaundiced hue, highly suggestive to the experienced clinician. A marked leucocytosis may be present, but it is often absent in uncomplicated cases. Later on, spasmodic cough, basal dulness, tubular breathing, or even amœbæ in the sputum indicate invasion of the lung. A fatal pericarditis, peritonitis or pyæmia may ensue.

Diagnosis.—The history of the case, the symptoms and the signs may in themselves be sufficient to make the diagnosis clear. The failure of quinine is an important guide, for an intermittent fever which does not answer to quinine is probably not malarial. The blood should be examined for the malarial organism, but even when it is found, the possibility of both diseases (malaria and abscess) being present should not be lost sight of. Exploratory aspiration with a not too small needle should always be undertaken in suspected cases. Failing an area of posterior dulness, the needle should be entered in the seventh interspace in the mid-axillary line, and the puncture must be repeated again and again. The patient should be put under ether and the various punctures made, so as to, as far as possible, explore every part of the liver before desisting. Little danger is to be apprehended from the procedure, even when a very large number of punctures have to be made.

Prognosis.—It is always serious. The mortality is large, perhaps about 50 per cent., even at the present time, but it is greatly lessened from what it was a comparatively small number of years ago, owing to greater skill in the diagnosis and surgical treatment of the disease.

Treatment.—Tropical abscess is generally single, and its treatment by drainage is often successful, although sometimes it may open into some neighbouring viscus, and may become evacuated without setting up peritonitis, pleurisy or other serious inflammatory condition. As regards drugs, quinine should be freely administered where the temperature tends to become hectic.

Symptoms.—2. OF THE PYÆMIC ABSCESS OR OTHER MULTIPLE ABSCESS.—They are those of pyæmia generally. The liver is usually enlarged. Pain is very often absent. A pronounced leucocytosis is often present. Emaciation and loss of strength gradually follow. A fatal issue supervenes sooner or later.

Diagnosis.—The history of the cases, gall stones, enteritis, endocarditis, &c., will generally help in establishing a diagnosis when the enlargement of the liver and general *pyæmic symptoms* appear.

CHRONIC INFLAMMATION OF THE LIVER— CHRONIC HEPATITIS, FIBROSIS OR CIRRHOSIS.

A large number of toxic agents, of mild strength and prolonged action, may bring about changes in the liver. In it, as in other glandular organs, while the primary action of the irritant may be either upon the liver cells or upon the fibrous tissue framework, the morbid changes produced come in time to present much the same appear-

ances, and an increase in the interstitial fibrous tissue is their ruling feature, hence they are spoken of as fibroses of the liver, and since in the majority of such cases the whole organ is of a pronounced yellow colour, the term cirrhosis was adopted by Laennec.

Etiology.—Of the many agents brought forward with varying degrees of proof as the causes of cirrhosis, the foremost places are taken by (1) *alcohol* and (2) *syphilis*, and most of the others can be arranged alongside either the one or the other. The former heads a toxic group, including all agents likely to lead to faulty metabolism, *e.g.*, alcohol, lead, gout, &c., and the latter an infectious group, including all poisons of microbic origin, such as syphilis, malaria, typhoid and other specific fevers, as well as, in all probability, some non-specific inflammations. Theoretically, these two groups are easily distinguished, but practically they are not so distinct. In many cases both groups are probably at work. The liver is constantly receiving germs from the intestine, which are destroyed by the natural bactericidal powers possessed by the liver in the healthy state. The faulty metabolism of alcoholism, lead, gout, &c., may so impair its powers that these germs, instead of being destroyed, flourish and help either in the degeneration and destruction of hepatic cells or in the formation of fibrous tissue.

Morbid Anatomy.—Many varieties of cirrheses have been described under names expressing their chief characteristics, such as *atrophic*, *hypertrophic*, *fatty*, *biliary* and *syphilitic*. In the atrophic variety the newly formed fibrous tissue chiefly surrounds groups of lobules, hence it is also called *polylobular*; in the *hypertrophic* it chiefly surrounds individual lobules, hence also the term *monolobular*; in the syphilitic it penetrates between the hepatic cells, hence the term *pericellular*; in the *fatty* there is an immense

amount of fatty change in the hepatic cells, and in the *biliary* there is a striking increase in the biliary channels. The relations of the fatty to the atrophic variety are so close that the former may well be regarded as a sub-variety of the latter, and the same may be said of the biliary and hypertrophic. This reduces the chief varieties of cirrhoses to three—the atrophic of Laennec, the hypertrophic of Hanot, and the syphilitic. The classical teaching of Laennec that prolonged over-indulgence in alcohol in all its forms (gin being the favourite in earlier times, hence the term *gin drinker's liver*) led to the production of a small and atrophied liver, whose surfaces showed a number of small, irregularly-sized projections, somewhat resembling nails in the soles of a boot (hence the term *hobnailed liver*), has remained undisputed until recent times. Hanot's hypertrophic cirrhosis seemed to be altogether a different disease. Its causation, when not completely obscure, seemed to be due to some infectious process rather than to alcohol. The liver is hypertrophied, sometimes greatly, and presents a comparatively smooth surface. The newly-formed fibrous tissue is monolobular rather than polylobular, and is far richer in new biliary channels. Combined with these etiological and histological differences there appear to be well marked clinical distinctions between the two, of which the chief are the *presence of jaundice*, sometimes slight, sometimes severe, and the *absence of ascites* in the latter. In atrophic cirrhosis it is the reverse, ascites being common, and jaundice absent or slight, while enlargement of the spleen is not usually the marked feature it is in the hypertrophic form. More extended observations have greatly shaken these views. The alcoholic liver—the atrophic liver of Laennec—is often, if not always, enlarged in its earlier stages, and may remain enlarged till the end. It has been shown, in short, that cases occur illustrating intermediate links, both histologically and clinically, between the classical atrophic and hypertrophic forms, and the

view is gradually gaining ground that these two forms of cirrhoses are one and the same disease. The essential morbid change is the increase of fibrous tissue within the liver. It begins as an increase of the fibrous tissue of the portal tracts (Glisson's capsule) around the median branches of the portal vein in many cases. It extends thence along the branches of these veins, but is not confined to them or to the periphery of the lobules, for it forms bands which may pass right through the substance of a lobule. At the same time it is also true that in the typical atrophic forms it shows little tendency to penetrate between the cells at the margin of the lobules, whereas it does so to a considerable extent in the typical hypertrophic forms, and is a characteristic feature of the hereditary syphilitic variety. The bands of fibrous tissue above mentioned penetrate the liver substance in every direction, and in time show as a network of white, dense strands of varying thickness, in the meshes of which lie the masses of liver cells, which have a pale white or yellow (frequent) or yellowish-green colour from bile staining. In their progress the fibrous bands extend outwards to the general capsule over the surface of the liver, and becoming adherent to it cause it to become thickened and retracted below the surface at these points. The little portions of liver tissue at the surface, which intervene between these bands, project beyond the thickened adherent parts of the capsule, which forms a sulcus around them. They constitute the nodular appearance of the surface. The size of these nodules may be merely the mechanical result of the retraction of the capsule and fibrous tissue surrounding them, but they may be in part due to an actual new formation of liver cells within them as a compensatory process. Evidence of regeneration may be seen within the liver cells of these nodules. The bands are mostly composed of well-formed, dense fibrous tissue, but clusters of round cells may be seen in places. These are more

frequent in the hypertrophic than in the atrophic forms, and may indicate more actively growing areas. The cells consist partly of leucocytes and partly of connective tissue cells. The presence of short parallel rows of cubical or somewhat flattened cells is often seen within the fibrous tissue, particularly near the margins of the hepatic lobules. They are usually, though not always, seen much more frequently and in greater numbers in hypertrophic than in atrophic forms, and are suggestive of newly formed bile capillaries. In many cases they can be injected from the bile ducts, and can even be seen to spring from them. They may signify an attempt at regeneration of liver cells, but this does not explain them all, for some are probably liver cells in the process of disappearing—a reversionary degeneration; while some may be intra-lobular bile capillaries whose surrounding liver cells] have completely disappeared.

Another feature of interest is the presence of newly-formed, wide, thin-walled blood-vessels, often very numerous, within the fibrous bands. They can be injected from the hepatic artery. On the other hand, the branches of the portal vein are more or less occluded by the pressure of the surrounding fibrous tissue. The liver cells may be fatty, granular, pigmented, or show little change. They are often flattened at the margins of the lobules from pressure by the fibrous tissue. Small collections of large liver cells, staining deeply, may be seen in places. They have been thought to be cells of new formation—an attempt at repair. The obstruction to the portal circulation is of great importance. It leads to the formation of various venous anastomoses. The accessory portal system of Sappey plays a principal part therein. It consists in anastomoses of veins in the round and suspensory ligaments with the epigastric and mammary veins, and of branches about the gall bladder, the gastro-epiploic omentum, and in the suspensory ligament with the dia-

phragmatic veins. In the first of these two sets many of the anastomosing branches lie in the skin around the umbilicus, forming a network of dilated veins, the so-called caput medusæ. When present it is a help in diagnosis. A third set of anastomoses occurs between the gastric and œsophageal veins (the latter are often varicose and the cause of hæmatemesis). A fourth set between the hæmorrhoidal and inferior mesenteric veins may explain the frequency of piles in cirrhosis.

Another important anastomosis is between the veins of Retzius and the inferior vena cava and its branches. The veins of Retzius include those which unite the radicles of the portal veins in the intestines and mesentery and the retroperitoneal veins with the inferior vena cava. These various anastomoses may suffice to compensate for the obstructed circulation through the portal veins within the liver. The occurrence of ascites, which is frequent, is generally attributed to the failure of this compensating circulation, but it is extremely doubtful if this be its true or only explanation. It is in all probability largely due to a toxic condition of the blood. The enlargement of the spleen, which is usually present, though varying greatly in degree, is also to be regarded more as a reaction to this toxicity than as caused by the venous congestion. The congestion and chronic catarrh of the intestine is attributable to both causes. The pancreas and kidneys may show interstitial change. The heart often shows fatty change, and tubercular disease of the chest or abdomen follows in more than 25 per cent. of all cases. Cirrhosis of the liver may therefore be regarded as a general systemic disease with marked hepatic fibrosis and inadequacy.

Syphilitic Cirrhosis.—It is generally held that acquired syphilis may produce a cirrhosis indistinguishable from that described above, but this must not be regarded as proved, for these cases may be the result of the combination of syphilis and alcohol. The characteristic form of acquired

syphilitic cirrhosis is a very irregular one, consisting in irregular-branching masses of fibrous tissue dipping into the organ from the capsule or situated within its substance, and often dividing the liver up into large and very irregular-sized lobes. Gummata are often present within these bands, and it is possible that the other bands in which no gumma is seen are really cicatrices which mark the sites of gummata which have disappeared.

Hereditary Syphilis.—This form of cirrhosis is more markedly intra-lobular than any other. It is often spoken of as intra-cellular. There is an increase of fibrous tissue in the portal tracts as well. It is met with in infants who are the victims of hereditary syphilis, particularly in those who are still-born. The liver may show no change macroscopically and yet be found to be markedly affected when examined under the microscope, or it may be obviously enlarged and firmer than normal. Its colour varies from a grey to a yellow or brownish-red hue. Distinct fibrous bands are rarely visible owing to the fineness and diffuse distribution of the fibrous tissue, but miliary gummata are often to be seen as little white points, like minute pin heads. When larger, the centre is usually yellow, but large gummata are rarely met with. Microscopically, the fibrous tissue consists mostly of fine fibres containing few cells running into the lobule, along the walls of the capillaries and otherwise penetrating between the individual liver cells and groups of cells. The lobular appearance so characteristic of hepatic tissue becomes lost, and single liver cells and little groups of liver cells, variously altered in structure, lying in a dense finely-fibrous tissue, take the place of the typical hepatic lobules. There may be little collections of round cells here and there, particularly in the portal tracts, and the gummata, when present, are seen as collections of cells degenerated towards the centre but showing round and irregularly-shaped cells towards the periphery.

Symptoms.—They are very variable and may never be sufficiently marked to lead to the detection of the disease during life. A certain proportion of cirrhoses are discovered accidentally on the post-mortem table in cases dying of quite different diseases. The early symptoms are those of dyspepsia and irregularity of the bowels or of chronic alcoholism. Hæmatemesis sometimes occurs early. It may be copious and repeated frequently, though it rarely proves fatal. Its source is the engorged œsophageal or gastric veins, either through a diapedesis or an actual rupture. Melæna often accompanies hæmatemesis, but may be present without it and may escape notice. It may, in the absence of hæmatemesis, take origin in the congested intestinal mucous membrane. Other symptoms of retarded portal circulation, such as enlargement of the spleen and of some of the cutaneous veins, piles and ascites, soon follow in most cases. Epistaxis is frequent. The symptoms are generally slight so long as the collateral circulation continues to be effective. The toxic influence rarely makes itself evident during this period, except in some of the hypertrophic cases, in which the fever (102° to 104°F.), the jaundice (often very severe), and the delirium, stupor or coma which usher in the fatal termination are striking manifestations thereof; but even in ordinary cases some evidence of its presence may be obtained in a slight irregular pyrexia (100° to 102°F.), slight jaundice and nervous symptoms. When the circulation begins to fail, the leading features of cirrhosis either begin to appear or become more evident. They are the *size of the liver* and of the *spleen*, *ascites*, *jaundice*, *dilated veins in the skin of the abdomen*, *piles*, the *hepatic facies*, and *progressive weakness*. Some of these symptoms have already received sufficient notice, but a few words must be added to the others.

The *size of the liver* varies greatly. It may be almost a half of or even twice its normal size. It is probably always enlarged to begin with, and, though it may after-

wards diminish progressively in size, the ultimate production of a smaller liver than normal is probably the exception and not the rule. In some cases the enlargement is always great and tends to increase rather than diminish. Whether enlarged or contracted, the clinical symptoms and course are much the same (Foxwell). Enlarged livers are generally easy to make out by palpation and percussion, contracted ones often difficult or impossible, particularly if there be much ascites.

Enlargement of the spleen great enough to be palpable is present in about 75 per cent. of all cases. It is usually most marked in the hypertrophic forms.

The *ascites* and *jaundice*.—The smaller the liver the more marked the ascites, the larger the liver the more marked the jaundice, is true as a general statement only, for there are many instances in which a large liver is associated with much ascites and little jaundice, and others in which both are well marked. The amount of fluid in the peritoneal sac varies from a few to many pints. It consists of a pale straw-coloured, limpid fluid which, although it may show some soft fibrinous clots, does not coagulate spontaneously to any extent after withdrawal. If allowed to accumulate within the peritoneal sac it causes disturbances in the respiratory and cardiac functions, *e.g.*, breathlessness, cough and palpitation, and also increases the general debility. Jaundice is not present in all cases, perhaps in little more than half. It is slight in cases with contracted livers and usually great in cases with enlarged ones. It is probably caused by catarrh of the biliary duodenal orifice or of the bile channels and by inspissation of the bile, the result of toxic action and congestion.

The *hepatic facies*.—The face gradually assumes a very characteristic appearance, upon which many of the older physicians placed great reliance. The eyes are sunken, the conjunctivæ watery and often jaundiced, the face thin,

the complexion muddy or jaundiced, and the nose and cheeks show distended venules.

There is progressive loss of flesh and weakness, with increasing shortness of breath and distaste for food. The case terminates by asthenia, or with coma or delirium, or with hæmorrhage or some intercurrent disorder such as kidney disease or tuberculosis. This description of the symptoms applies equally to the acquired but not to the hereditary syphilitic cirrhoses. In the latter, the infants usually show other prominent signs of syphilis, such as skin rashes, snuffles, bronchitis. They have a withered, old-mannish look. Unless there is jaundice and obvious hepatic enlargement, the hepatic cirrhosis may not show itself among the more prominent signs of disease in other organs.

Diagnosis.—This is impossible in the early stages when the symptoms are indefinite and there is no history of alcoholism. Later, when there is ascites, enlarged spleen, piles, and hæmatemesis, there is little room for doubt. The greatest difficulty lies in differentiating some cases of malignant disease of the liver from cases of hypertrophic cirrhosis, but the more rapid course followed by the former generally enables the distinction to be drawn. The possible co-existence of both diseases is to be remembered.

Prognosis.—It is bad, though favourable cases may live for years. So long as the compensatory circulation remains effective the patient may do well, but whenever jaundice or ascites or both become prominent the end is not far off. The patient rarely lives for a year after tapping has to be employed, though some few do comparatively well for longer periods after repeated and early tapping. Some of these favourable cases may be syphilitic.

Treatment.—In *alcoholic* cases, the alcohol should be stopped entirely. Where ascites is present much benefit

results from promoting free diuresis and giving hydragogue cathartics. Amongst the best purgatives are concentrated salines administered warm before breakfast, and many physicians find a course of Carlsbad salts or other saline aperient given daily helps greatly to reduce the dropsical effusion in such patients. An attempt should also be made to induce the patient to take dry diet, and salt should be eliminated from the food. Sooner or later, however, tapping is necessary to relieve the abdominal distension and consequent pressure on the diaphragm. A Southey's tube inserted carefully in the mid-abdominal line, between the umbilicus and the symphysis pubis, ensures the draining away of the fluid slowly and yet effectively. Before inserting the tube the bladder should be emptied, and while the fluid is being drawn off a binder should be applied to the abdomen, and this should be gradually tightened so as to make up for the reduction of pressure. In certain cases surgical interference has been tried for the purpose of establishing anastomosis between the portal and general circulation. This may be attempted either by stitching the omentum to the abdominal wall or endeavouring by scratching the peritoneal covering of the liver, or by the introduction of sutures, to make the liver become adherent to the parietal peritoneum. Dyspepsia, from which the patient often suffers, requires treatment; hæmatemesis is beneficial, and, unless excessive, need not be arrested; and pain should be relieved by fomentations or linseed poultices. Potassium iodide has been recommended and may be tried in short courses for a few weeks at a time. In *hypertrophic* cirrhosis it is difficult to treat the toxæmia present by any satisfactory method. In *syphilitic* cases, whether hereditary or acquired, potassium iodide should be administered.

YELLOW ATROPHY—ICTERUS GRAVIS—
MALIGNANT JAUNDICE.

This rare disease is usually acute, terminating fatally in about a fortnight after its onset, but a certain number of cases are subacute, lasting for about four or five weeks or sometimes for longer periods.

Etiology.—No certain cause has so far been discovered, but there are considerable grounds for placing it among the infective processes. Its pathology, so far as it is known, almost justifies it being included under acute inflammations of the liver, but its clinical picture and the histological appearances in the more chronic forms, resembling as they do some forms of cirrhoses, make it more convenient to place it by itself immediately after hepatic inflammations. Putting aside pregnant women, who seem specially liable to the disease, it occurs with about equal frequency in both sexes. A wound in the skin or gastro-intestinal tract has been present in some of the cases, and may have acted as the primary source of infection, but an actual wound may not be necessary, particularly in the bowel. Bacteria, chiefly the colon bacillus and other intestinal forms, have been found in the diseased parts of the liver, but their relationship thereto has not been determined. Acute phosphorus poisoning produces changes very similar to those of acute yellow atrophy.

Morbid Anatomy.—The liver is at first enlarged but its size soon diminishes, until at death it is considerably smaller than normal and may weigh only 30 ounces, or even less. It is thin and shrunken looking, and its capsule is wrinkled. On section, it presents in most cases a mottled yellow and red appearance, though in some there

is a considerable admixture of green. The latter is due to bile staining, but the red and yellow areas represent two stages in the diseased process, the former being more advanced than the latter. The essential part of the change is a necrosis of the liver cells accompanied by a dilatation of the hepatic capillaries with hæmorrhages and an increase in the interstitial tissue. The liver cells become swollen, granular, fatty, lose their staining powers, and begin to break down. The cells are stained yellow by bile pigment. This constitutes the yellow patches. At first there may be little or no congestion of the intra-lobular capillaries, though there may be an increase of leucocytes therein, and even outside them among the liver cells. The portal tracts are crowded with rounded cells, many of which are leucocytes and others proliferating connective tissue cells. As the disease progresses the liver cells break down more completely, the capillary congestion and leucocyte invasion increase, and numerous hæmorrhages appear. Leucin and tyrosin may be seen in the liver cells, and more abundantly in the debris resulting from their death. The yellow patch thus passes into the red patch, which usually shows no recognisable liver cells, or only a trace thereof, their place being taken by debris in which there are many fat globules, many leucin and tyrosin crystals, hæmorrhages, leucocytes and dilated capillaries. The disease begins at the periphery of the lobule and quickly creeps towards its centre, so that the lobular structure soon becomes lost. In the more prolonged cases (the subacute or somewhat chronic ones) the necrosis of the liver cells is not so extensive. It may affect the peripheral or an irregular part only of a lobule in which a fine fibro-cellular or fibrous tissue (according to the chronicity of the process) has taken the place of the vanished liver cells, and in this fibrous tissue many cells arranged like young bile ducts in parallel rows are usually to be seen, thus presenting a picture

very like that of some forms of cirrhosis. They are held by some to signify a degenerative or reversion process, by others a regenerative process. Both views are probably correct, and there is further evidence of regeneration in the form of the occasional occurrence of mitotic figures within scattered patches of swollen liver cells. There are some signs of inflammation in the smaller bile ducts, and Hunter holds that this is the essential morbid change in the disease. He thinks that the poison acts chiefly upon the finer bile ducts, resulting in an angiocholitis which prevents the free flow of bile from the liver and produces the jaundice and other local and general toxic symptoms of the disease. The other cellular organs, *e.g.*, the kidney and heart, show granular and fatty degeneration, and all the organs and tissues of the body are bile stained and show many hæmorrhages.

Symptoms.—It usually begins, like ordinary simple or catarrhal jaundice, with signs of gastro-intestinal inflammation. There may be nothing to distinguish it from ordinary jaundice for a week or more, but sometimes there is enlargement and tenderness of the liver. The bilious vomiting which occurs at first is not characteristic, but presently the gastro-intestinal symptoms become more severe. The jaundice increases, the vomiting becomes more frequent, and towards the end the bilious vomit usually becomes coffee-ground-like from the admixture with blood. Nervous symptoms of two types appear, sometimes very suddenly, either drowsiness, sinking into coma, or headache, delirium and convulsions, or a combination of the two types. Constipation is frequent, the stools being often clay-coloured. The liver dulness gradually diminishes and may disappear. The urine contains leucin and tyrosin and diminished urea in most cases, albumen and tube casts in some, and an increase of nitrogen in the form of ammonia in others. The tempera-

ture is normal or even subnormal till near the end, when it may rise as high as 107°F . The skin is hyperæsthetic, and the weight of the bed-clothes, or even a slight touch, may cause great distress. Hæmorrhages into the skin and mucous membranes are frequent. Pregnant women usually abort. Most cases end fatally within a few days after severe intestinal or nervous symptoms set in.

Diagnosis.—In typical cases, in which the jaundice increases, the liver dulness decreases, the urine contains leucin and tyrosin and nervous symptoms come on, there is no difficulty. It most resembles acute phosphorus poisoning, which may be distinguished from it by the garlicky odour and luminosity of the vomit in the dark, by the greater size of the liver, and by the rare presence of leucin and tyrosin in the urine.

Treatment.—Unfortunately we do not know any effective treatment. Palliative measures should be adopted for the relief of the vomiting and constipation, and it is certainly desirable to administer alkaline injections, either by a vein or per rectum, but nothing has so far proved really efficacious.

NEW GROWTHS.

Simple tumors, such as angioma and adenoma, occur, but they rarely reach a size large enough to become of clinical importance. Malignant tumors, on the other hand, are common, cancers occurring far more frequently than sarcomas, both being commonly secondary, the former to cancer of the stomach or rectum, &c., and the latter to sarcoma of the choroid or skin.

Morbid Anatomy.—Primary cancer of the liver is rare. It is met with in more than one form. There is (1) the massive form, in which there is one large cancerous mass with one or two smaller ones near it; (2) the nodular form, in which the primary growth reaches no great size and gives rise to numerous nodules like itself throughout the liver; (3) the cirrhotic form, which can only be distinguished microscopically from an ordinary cirrhosis by finding cancer cells imbedded in the fibrous tissue; (4) the biliary form, which originates in the walls of the small intra-hepatic bile channels and forms numerous small cancerous nodules everywhere throughout the liver; and (5) the hypernephrotic form arising in particles of the suprarenal body included within the liver. The size of the liver is increased, often greatly, in the first form, moderately in the second, fourth and fifth, and actually diminished in the third, and sometimes in the fourth form also. Secondary cancer forms over 95 per cent. of all cases, and shows a varying number of nodules, generally numerous, some of which may be large and some small. Large nodules often undergo fatty degeneration and softening in the centre. This leads to contraction of the central part of the nodule, so that if situated on the surface of the organ, the nodule is distinctly umbilicated, so much so, that this can be felt through the abdominal wall in life. Primary sarcoma forms large masses, white and soft, and frequently necrotic in places and much mixed with blood clot. The organ is much enlarged. Secondary sarcoma forms numerous smaller masses, not unlike cancer, everywhere throughout the organ.

Symptoms.—There are usually none of a definite character until the liver begins to be noticeably enlarged. Pain and tenderness in the hepatic region is often, though not always, present. As the liver enlargement increases, it may be found to be nodular and irregular, and umbili-

cation may be detected. The general aspect of the patient is pale and sallow and there is progressive emaciation. Jaundice often appears towards the later stages, and sometimes also ascites, particularly in the cirrhotic forms, but it is frequently absent or slight in other varieties. The spleen is rarely enlarged. Gastro-intestinal symptoms, such as nausea, vomiting and hæmatemesis, may appear. An irregular and moderate pyrexia is frequent. The patient gets steadily weaker and usually dies of asthenia or cholæmia in from three to fifteen months, rarely more.

Treatment.—The treatment consists in relieving abdominal distension by tapping ascitic effusions when necessary, soothing pain by the use of fomentations or poultices, and by treating other symptoms as they arise. Surgical interference is in most cases absolutely hopeless, and even where a single growth is present and could be removed, the operation is a dangerous one and success is very problematical.

PARASITES.

Hydatid disease is the only parasitic disease of importance in the liver. It is conveniently described immediately after new growths, as clinically it is often difficult to distinguish it from a malignant tumor. The liver is the most frequent site of hydatids, the peritoneum coming next.

Morbid Anatomy.—The cysts vary in number (one being perhaps the commonest) and in size. They may be quite small or reach a large size. They lie imbedded in the liver, from which they are separated by a fibrous zone of inflammatory origin. The true cyst wall, consisting of ectocyst and endocyst, lies inside this capsule. It contains the fluid and the scolices. Usually the primary cyst contains

secondary or daughter cysts, called brood capsules, *vide* page 259.

Symptoms are usually absent for a long time, sometimes throughout life. When they appear they are generally due to pressure and resemble in part those of other tumors. Hydatids are rarely painful, and they are not accompanied by loss of flesh or emaciation. They are usually softer than cancers and run a much more prolonged course. The hydatid thrill may be felt. The cysts may be punctured, when the chemical and microscopic examination of the fluid may set all doubt at rest.

Treatment.—Hydatid cysts should be tapped, and often without further treatment cure is effected. It is very unwise to inject an antiseptic into the cyst or to endeavour to destroy the embryos within by electrolysis. Should suppuration occur in the cyst, the wall should be stitched to the abdominal wound and free drainage carried out.

VIII.—*DISEASES OF THE GALL BLADDER AND BILE DUCTS.*

GALL STONES.

Etiology.—The cause: underlying their formation vary a good deal, at any rate those which predispose to their formation do, though their actual cause is in all probability one or other of the inflammatory germs which invade the bile ducts and gall bladder from the intestine, such as the pyogenic cocci, the diplococcus lanceolatus, the colon and typhoid bacilli. They have often occurred after

typhoid or other infectious fever. They have been experimentally produced in animals by injecting germs into the gall bladder. The predisposing causes are age (most common about forty), sex (three-fourths of all cases occur in women), good living, and sedentary habits of life, all conditions in short which favour stagnation of bile. The gall bladder is the chief seat of their formation, though some are formed in the bile ducts within the liver. The germs probably act by setting up a catarrh of the mucous membrane which leads to the desquamation of epithelial cells and to the excessive formation of cholesterin, mucus and lime salts. The epithelial debris and bile pigments form a nucleus, around which the mucus, lime salts and cholesterin are deposited. Bacteria have also been found in the nucleus.

Morbid Anatomy.—*A. Physical characters of gall stones.*
—Their number, shape, size, colour, consistence, weight and composition have all to be noted. They are usually *multiple*, a score or more being common. The more numerous they are, the smaller the individual stones are likely to be. When moderately numerous—two or three score—they vary in size from a small pea to a bean, and have a polygonal form with smooth faces from mutual pressure. Exceptionally there is only one stone and then it is oval or round, smooth or nodulated like a mulberry, and may be of large size. Their colour is brown or blackish-brown on the outside, but pale or colourless within, usually showing concentric lamination and some radiating lines. The outer crust is a little harder than the interior, which cuts easily and has a greasy feel. They are extremely light for their size when compared with urinary calculi. This is due to their consisting for the most part of cholesterin. Their other ingredients are lime salts, bile pigment and mucus, and though the proportion of these three ingredients varies, the cholesterin is always most abundant, forming mostly 70

to 80 per cent. Those formed in the bile ducts within the liver constitute an exception in so far as they are mostly composed of bile pigment and lime salts with little or no cholesterin. They differ also in being very small, irregular in shape, and very dark in colour. They are sometimes known as gall sand.

B. Effects upon the gall bladder, ducts and surrounding tissues.—They may be arranged in two groups, according as they follow gall stones which (1) remain within the gall bladder, or (2) travel downwards to the intestine. In the first group their effects may be so slight as to be negligible. Gall stones are often accidentally discovered in considerable numbers after death, in cases in which their presence was unsuspected during life, and the gall bladder walls show very little change. They may, however, cause a simple catarrh of a comparatively mild character, which has little result beyond causing the bile to become paler, more viscid and ropery from its greater admixture with mucus, or they may cause a more severe catarrh, a definite *cholecystitis* of a simple or suppurative character. The latter is known as an empyema of the gall bladder. The inflammation may spread by rupture or without it to the peritoneum, skin or intestine, or, if it subsides without spreading, it may be followed by much contraction and calcareous infiltration of the walls. Severe cholecystitis which subsides is generally followed by much thickening of the wall and adhesion to the tissues around, as well as shrinking, contraction and deformity of the gall bladder itself. It is not the gall stones but the bacteria which invade the gall bladder from the intestine which produce these results. The gall stones owe their origin to the same bacteria and are merely incidents in the disease. Another condition which is closely associated with gall stones is *cancer of the gall bladder*, for gall stones are present in nearly every case of cancer of the gall bladder, though it is also true that the great majority of cases of gall stones are not

followed by cancer. In the second group the gall stones may travel downwards and escape into the intestine. They usually cause pain (biliary colic) in their progress, and are also followed by catarrh, which generally causes a certain amount of jaundice. They may be arrested in their downward course, either in the cystic duct or in the common bile duct, causing complete or incomplete obstruction thereof. Obstruction of the cystic duct may be followed by *shrinking* or *dilatation* of the gall bladder. In the latter case it contains mucus mixed with bile or pus, or both in the acute cases, and clear mucus alone in the chronic cases. It is frequently large enough to be palpable as a pear-shaped or gourd-like swelling below the ribs, occasionally so large as almost to fill the abdomen. Shrinking may follow dilatation. Obstruction of the common bile duct may be complete and permanent or incomplete. In the former it causes dilatation of the main hepatic duct and its branches within the liver without much or any inflammatory change, but with deep and enduring jaundice; in the latter similar changes are produced, but in certain cases they are accompanied by thickening and roughening of the walls of the dilated ducts, and sometimes by suppuration within their lumen and in the liver substance around them, constituting abscesses. Incomplete obstruction allows of the passage of germs from the intestine into the ducts above the stone. The calculi may produce a varying degree of inflammation of the walls of the ducts, sometimes so slight as to be hardly noticeable, at other times severe and suppurative, though it is to be remembered that stones may exist for years in the ducts and imperfectly block them without suppuration following. There is greater enlargement of the liver in the suppurative cases. Jaundice is generally present, but its intensity varies at different times. The commonest site of obstruction of the common bile duct by a gall stone is the ampulla of Vater. In this case it will interfere with the emptying of the pancreatic as well

as of the bile duct, and may be followed by dilatation, with or without inflammation, of both ducts and their branches. A stone encysted in the gall bladder, or impacted in the cystic or in the common bile duct may ulcerate through into the abdominal cavity, into the duodenum, the colon or other neighbouring structure. A gall stone may reach the duodenum and cause intestinal obstruction lower down, though it rarely does so.

Symptoms.—They are very variable, according to the site of the gall-stone and the degree of attendant inflammation. As already mentioned, many gall stones may exist for long periods in the gall bladder without giving rise to any symptoms, and when small enough they may even migrate to the intestine without doing so. On the other hand, they frequently cause *pain* and *fever*. The pain of biliary colic, *i.e.*, caused by the migrating stone, is sudden and violent. It radiates from the hepatic region in various directions—laterally, backwards and upwards—towards the right shoulder. The patient often rolls about in agony, perspires freely, and vomits frequently. The pain subsides with the escape of the stone into the bowel. The attack varies in duration, lasting for a few hours or several days to a week or more. The pain caused by a stationary stone is much less severe. When present it is usually felt in the hepatic region and is increased by palpation. The fever varies in degree, and may reach 103°F. or even 105°F. It subsides with the escape of the stone into the intestine. In other cases it remains and may be intermittent, particularly when the stone is impacted in the ampulla of Vater. In the suppurative cases it usually shows the zigzag course characteristic of pyæmia. In addition to the pain, fever, sweating and vomiting, there are shiverings or rigors, feeble action of the heart, and jaundice. In the biliary colic the jaundice—usually comes on in the course of a few hours after

the attack commences, and subsides rapidly after it ceases. It is generally slight. In the case of a stone impacted in the cystic duct it is absent, while it is present in cases in which the impaction is in the common bile duct. When the impaction is complete, the jaundice is permanent and steadily increases, when incomplete it is variable and deepens with each paroxysm.

Diagnosis.—It is generally easy in “biliary colic,” particularly if jaundice follows the attack and if there be a history of previous attacks. The stools should be rendered liquid with weak carbolic and strained through a small meshed sieve and gall stones looked for. Failure to find them does not negative the diagnosis. Biliary colic is liable to be confounded with appendicitis, renal colic, lead colic or gastralgia. In appendicitis the pain is generally greatest in the cæcal region. When tenderness and swelling appear the separation becomes easy. In renal colic the pain is lower than in biliary colic, and it radiates downwards. In lead colic there is no pyrexia and a blue line may be seen on the gums. In gastralgia there is less fever. Obstruction of the cystic duct is distinguished from obstruction of the common bile duct by the absence of jaundice and the large size of the gall bladder. Obstruction of the common bile duct is diagnosed by the jaundice, the history of gall stones, and by the other symptoms characteristic of simple or suppurative inflammation of the bile ducts, viz., the rigors, the intermittent fever, and the sweating. Attacks may recur again and again for years, symptoms being slight or absent in the intervals, but a varying degree of jaundice usually persists. The suppurative variety is distinguished by the fever being greater, remittent rather than intermittent, by the jaundice being less intense and less variable, by the greater size of the liver, and by the general resemblance to other pyæmias or septicæmias.

Treatment.—For an attack of biliary colic try the effect of a hot bath to relax the spasm and to assist, if possible, the stone or stones in their passage towards the duodenum. It is generally necessary to give a hypodermic injection of morphia, and in very severe cases even to put the patient under the influence of chloroform. Large draughts of an alkaline mineral water and a dose of a saline cathartic will be found helpful. It is imprudent to endeavour to assist the stone in its passage downwards by massage. Between the attacks of biliary colic an attempt should be made to prevent the formation of more calculi, and no remedy, in the opinion of the writer, is of such value as sodium phosphate in 1 drachm doses thrice daily. This salt appears to render the bile less viscid. There is considerable difference of opinion with regard to the possibility of dissolving calculi actually in the gall bladder. The administration of salad oil by the mouth has been strongly commended, but it is impossible to believe that any of the oil can ever find its way up the ductus choledochus and along the cystic duct into the gall bladder.

Where there is much jaundice associated with an attack of gall stones, or due to a gall stone blocking the common bile duct, itching of the skin may require treatment. A good solution with which the skin may be sponged consists of chloral (2 drachms), liquor ammonii acetatis (10 ounces), with equal parts of water; or a solution of bicarbonate of soda may be used in a similar way. In other cases a hypodermic injection of pilocarpine ($\frac{1}{12}$ th to $\frac{1}{8}$ th grain) is promptly beneficial. When permanent jaundice results from gall stones, or it is evident that one or more gall stones are present, an attempt should be made to diagnose the position of the stones. When they are in the gall bladder or cystic duct they may be removed by cholecystotomy; if in the liver, preliminary exploration with a needle is warranted. The treatment (by purely medical means) of gall stones which cannot, or do not, find their

way downward into the intestines should not be persisted with for too long a time, because suppuration may occur, and occasionally ulceration of the stones takes place into the intestine or even into the peritoneal cavity. An important point for the surgeon to remember, in all cases where there is permanent jaundice, is the increased tendency to hæmorrhage in operative procedures.

INFLAMMATION OF THE GALL BLADDER— CHOLECYSTITIS.

Etiology.—This is caused by germs which reach the gall bladder from the intestine through the bile ducts. The vast majority of cases are associated with gall stones.

Morbid Anatomy.—The gall bladder is generally more or less distended with viscid mucus forming a pyriform tumor attached to the under surface of the liver. Its outer surface is smooth, but its inner surface may be thickened, rough and ulcerated. In suppurative cases it contains a varying amount of pus.

Symptoms.—When the inflammation is simple they closely resemble those of biliary colic with the symptoms of peritonitis superadded; when it is suppurative they may be much the same but somewhat intensified or resemble those of pyæmia. The presence of a pear-shaped tumor, tender to the touch, and the comparative slightness or absence of jaundice, are important diagnostic points.

Treatment.—Until the surgeon can be called in, ice should be applied over the region of the gall bladder. Aspiration has been recommended, but it is better to stitch the gall bladder to the edge of the abdominal wound and

drain freely, removing at the same time any gall stones which may be present.

INFLAMMATION OF THE BILE DUCTS— ANGIOCHOLITIS—CHOLANGITIS.

It may be simple or suppurative. In the simple form a distinction must be made between catarrh of the *large* and of the *small* bile ducts.

INFLAMMATION OF THE LARGE BILE DUCTS.

Etiology.—They are most frequently affected at the orifice of the bile duct into the duodenum, but may be attacked in any part of their course. Inflammation of the orifice or terminal part of the bile duct is usually caused by a catarrh of the stomach—a gastritis—spreading downwards into the duodenum. It is most frequently met with in young people, but may occur at any age. Inflammation of the larger trunks of the bile duct are almost always due to gall stones and have already been considered. Inflammation of the orifice of the bile duct constitutes ordinary *catarrhal jaundice*.

Morbid Anatomy.—The catarrh involves the orifice of the common bile duct and probably does not spread any distance into it. It leads to the formation of mucus, a plug of which, or the swollen lining cells, or both together, may completely block the lumen for a time.

Symptoms.—In many cases there are no noticeable symptoms until jaundice comes on, and the patient may appear to be in good health—so-called biliousness in mild cases. In other cases there are distinct signs of gastritis or duodenitis. Fever is either slight or absent, the stools are

clay-coloured and the urine contains bile pigment. The gall bladder is rarely enlarged. The bright yellow tint of the jaundiced skin is characteristic. In mild cases the jaundice and other symptoms, if present, disappear within two weeks, in other cases they may persist for months. The average duration is about four to eight weeks. In the more chronic forms pruritus may be present and cause much distress. The pulse is usually slow (forty to thirty per minute), particularly in the early stages. Yellow vision may occur. Skin hæmorrhages are common, and this tendency to hæmorrhage is important in relation to operations. Nervous symptoms, such as mental depression, delirium, convulsions, or coma, may arise in prolonged cases. The nervous symptoms have been grouped under the term "cholæmia," and their method of production is not clearly understood. They are more common in the next group of jaundice cases—"the febrile or toxæmic," due to inflammation of the finer intra-hepatic bile ducts.

Treatment.—In mild cases of catarrhal jaundice give the stomach as much rest as possible, administer sedatives such as bismuth and hydrocyanic acid, and later calomel in 2 grain doses, twice daily, or some other hepatic stimulant. Limit the diet to milk. The itching due to jaundice should be relieved, if present, by sponging the skin with a solution of chloral (1 to 2 drachms) in 10 ounces of liquor ammonii acetatis—this mixture being diluted in an equal part of water.

INFLAMMATION OF THE MINUTE BILE DUCTS.

Etiology.—Many poisons during their elimination by the liver are capable of causing a mild inflammation of the minute bile ducts. The result of this catarrh is a swelling, and perhaps a desquamation of the lining epithelium, with an increased secretion of mucus. This renders the

bile more viscid and delays it in its passage downwards into the larger bile ducts, and thus allows of an unusual absorption into the blood, probably by the lymphatics, and a resulting jaundice. There is another feature in the action of these poisons which favours the production of jaundice, and that is an excessive destruction of the red blood cells of varying degrees caused by the poisons. The portal blood thus carries to the liver cells an increased amount of hæmoglobin, and consequently they form an increased amount of bile pigment. In past times two other opinions were held upon the production of the jaundice in these cases, one view held that the bile pigment was formed within the blood outside the liver from the liberated hæmoglobin following an increased hæmolysis of the red cells, hence the term "hæmatogenous jaundice." The other view, starting with the supposition that the bile was normally formed in the blood outside the liver, held that the liver cells had ceased to perform their function of extracting the bile from the blood, hence the term "jaundice by suppression." Both these views are untenable for the simple reason that bile is now known never to be formed within the blood, but only within the liver cells, and that no bile is formed if the liver be absent. Another view also held for some time, viz., that these diseases caused an excessive discharge of bile into the intestine, and a consequent excessive absorption therefrom ("jaundice from polycholia"), is also unsupported by any evidence. Jaundice is therefore always *intra-hepatic*, inasmuch as the bile pigment is a result of metabolism within the hepatic cells. In obstructive jaundice so-called the lesion is outside the liver (extra-hepatic) in the common bile duct, in toxæmic jaundice it is within the liver (intra-hepatic) in the minute hepatic ducts, and is also essentially an *obstructive* jaundice. In so far as the poison causes an increased hæmolysis, enabling the blood to bring an excessive supply of the materials to the liver cells, out of which they form bile and thus lead them to produce an

excess of bile, the term "hæmohepatogenous jaundice" is admissible. The poisons which act on the blood in this way may be grouped under two headings:—(1) Phosphorus, arsenic, chloroform, toluylendiamin, snake venom, &c.; (2) toxins produced by infectious diseases, *e.g.*, yellow fever, yellow atrophy, malaria, typhus fever, Weil's disease (a form of epidemic jaundice), pyæmia, &c.

Symptoms.—In addition to the symptoms of the primary disease, there are superadded those due to the consequences of the bile circulating in the blood, *viz.*, the yellow staining of the skin, mucous membranes and secretions, skin eruptions and pruritus, yellow vision, a bitter taste in the mouth, and cholæmia. In mild cases they may be very slight, but in well-marked cases they become distinctly noticeable, and are distinguished from the similar symptoms in obstructive jaundice by certain differences. For instance, there generally is fever, and consequently the slow pulse and respiration of obstructive jaundice are not observed. Cholæmia is much more likely to arise and to be more severe, and the stools are rarely clay-coloured, since the obstruction is partial and the increased hæmolysis increases the amount of bile pigment.

Treatment.—Treat any symptoms present. It is not always easy to discover the cause of the condition, but where a diagnosis can be made, it may be possible to do something to counteract the toxæmia so often present.

ICTERUS NEONATORUM.

This may be regarded as a special variety of simple inflammation of the bile ducts. It occurs in a severe and a mild form. The *severe form* may, as mentioned above, be syphilitic in origin and involve the terminal part of the common bile duct, or it may have some other cause,

for it cannot be said that all forms of congenital occlusion of the common bile ducts are of syphilitic origin. Of these other forms the most acute and fatal of all involves the finer bile ducts like toxæmic jaundice. It is believed to be caused by septic absorption through the umbilical cord causing phlebitis and jaundice. Hæmorrhage from the umbilical cord is very likely to occur and to prove very intractable. The *mild* form appears in a certain proportion of healthy infants, usually on the first or second day. It is most common in foundling hospitals, but it is not rare in private practice. The infant is otherwise healthy and the jaundice usually disappears within one to two weeks. It is never fatal. Its cause is not clear. Some think it is due to an excessive hæmolysis. Another view holds that there is obstruction to the flow of bile through the smaller bile ducts by an œdema of their surrounding tissue caused by dilatation of the branches of the portal veins. The sclerotics are much less tinged than the skin. The fæces usually show the normal colouration, though they may be clay-coloured. It has no apparent influence upon the health or nutrition of the infant, and hence has its chief interest in its scientific side.

SUPPURATIVE INFLAMMATION OF THE BILE DUCTS.

This form of inflammation is not confined to either the large or small ducts, as is the case in the simple form. It involves all the bile ducts, the larger and smaller alike.

Etiology.—It is most frequently seen after gall stones, less frequently after cancer of the bile duct or after lumbricoid worms. It is due to suppurative germs spreading upwards from the intestine. Occasionally it

spreads by direct extension from a suppurative pylephlebitis, and very rarely arises in acute general infections such as influenza.

Morbid Anatomy.—All the larger ducts, both outside and inside the liver, are dilated and their walls thickened. They contain a mixture of pus and bile. The gall bladder is usually similarly affected. There are numerous small abscesses around the small branches of the bile ducts within the liver. The gall bladder or common bile duct may have perforated into the peritoneal cavity, the portal vein, intestine or other neighbouring structure.

Symptoms.—The jaundice and other symptoms of the catarrhal form are soon shrouded by those of pyæmia. They are always severe and a diagnosis is difficult unless there is a history of gall stones. The jaundice, which is always present, although it may be slight, and the enlargement and tenderness of the liver are useful guides.

Treatment.—It is rarely possible to do much beyond treating the septicæmia, which is so often present. The administration of quinine, the application of ice for pain, and the use, perhaps somewhat experimentally, of anti-streptococcic serum are worth a trial. Where gall stones are present, or where there is any indication that surgical aid might save life, give the patient the benefit of the doubt and operate.



CANCER OF THE GALL BLADDER AND BILE DUCTS.

The gall bladder is far more commonly than the bile ducts the seat of cancer. The walls of the organ may be

merely irregularly thickened, but more usually a massive tumor, of a rounded shape, is formed within the gall bladder which can be felt during life attached to the lower border of the liver and extending downwards towards the umbilicus. Histologically it is composed of columnar or spheroidal cells. In most cases gall stones are present. They are believed to precede the cancer in the great majority of the cases, though they may be secondary to it in some of them. The cancer usually extends to the structures around the gall bladder in the hilum of the liver, forming a large growth which presses upon the bile duct and portal vein branches. It often radiates into the liver substances as well, and may form secondary detached nodules within it. Primary cancer of the common bile duct, on the other hand, is comparatively rare. It may grow in any part of the duct, but is most frequently seen in the duodenum around the biliary orifice and in the ampulla of Vater. In the former situation it usually forms a larger tumor than in the latter, but it is rare for either of them to reach a size large enough to be felt through the abdominal wall. In the ampulla of Vater it is usually of very small size, appearing merely as a slight thickening of the wall, not sufficient to close the lumen were it not for an intercurrent catarrh which is likely to arise at intervals. Primary cancer may also attack the intra-hepatic bile ducts, forming numerous small nodules everywhere throughout the liver substance. It is practically a cancer of the liver.

Symptoms.—The symptoms of cancer of the gall bladder are indefinite for a time. Pain, both paroxysmal and severe, in the region of the liver and radiating to the right shoulder is an early symptom. Anæmia and wasting gradually come on, but may in some cases never be prominent. Gastric symptoms are sometimes marked from the first. Jaundice comes on in most cases later in the disease.

There is often a history of gall stones. In its later stages, after the cancer can be felt, the course is much the same as that of cancer of the liver. Primary cancer of the bile ducts may be suspected from the early appearance of the jaundice. It is usually intense and persistent, but may vary in intensity from time to time. Interferences with pancreatic digestion and pancreatic function, *e g.*, diabetes, may come on.

Treatment.—The treatment can only be palliative, and at an early stage the use of morphia will give the patient much relief from the frequent and often constant pain.

STENOSIS AND OBSTRUCTION OF THE BILE DUCTS.

Etiology of Stenosis.—Inflammation is the usual cause, and it most frequently attacks the lower part of the common bile duct. Cancer is a rare cause. Inflammation is oftenest caused by gall stones, but worms or other foreign bodies entering from the intestine may set it up. Hereditary syphilis is another cause. The terminal part of the bile duct may thus be completely occluded. In some infants, the victims of hereditary syphilis, it has been found to be converted into a fibrous cord (one of the forms of *icterus neonatorum*).

Etiology of Obstruction.—Obstruction is most frequently caused by pressure from without. Cancer of the head of the pancreas is the commonest cause of pressure upon the terminal part of the duct, and cancer (secondary to stomach) of the lymphatic glands or of the gall bladder of the main part of the duct.

Symptoms.—Jaundice is the most striking of the symptoms. These are in themselves very much the same as those of chronic obstructive jaundice from any cause, and when the occlusion is incomplete and the primary disease permits of it, cases may live for several years. When it is complete, death follows.

IX.—*DISEASES OF THE PANCREAS.*

The pancreas secretes the most powerful of the digestive juices, and there is also considerable evidence in support of the view that it has another secretion—an internal one—interference with which leads to the production of diabetes. The physiological importance of the organ accordingly suggests that its diseases ought to make themselves known by characteristic symptoms; but this is not the case. Considerable structural changes may arise without producing any recognisable symptoms, and when they are produced they are not, either singly or combined, pathognomonic of pancreatic disease. At the best they are only suggestive thereof. The infectious fevers, and other wide-spread morbid states which produce granular and other degenerative changes in the glandular organs, must produce similar changes in the pancreas and interfere in some measure with its functions. Interferences with the digestive action of the pancreas are not easily recognised, because the salivary, gastric and intestinal juices may compensate for its deficiencies. When the interference is considerable, fat may be recognised in the fæces and the urine, and emaciation may come on rapidly. In the stools fat appears as an oily film on the surface of the fæces, as oily globules and masses, or as crystalline-looking material consisting of fatty acids and salts. The stools do not, however, always show fat in any of these forms, even in extensive disease

of the pancreas, and they may do so when the intestine and not the pancreas is the seat of the disease, *e.g.*, in any condition leading to a great diminution of the absorptive power of the intestine. Fat in the urine is rare, it may indicate pancreatic disease, but it occurs also in other diseases, *e.g.*, fat embolism, diabetes, &c. A diminution in the amount of indican in the urine is said to be also present. The presence of a considerable amount of undigested muscle fibre in the fæces is held by some to suggest deficiency in the pancreatic juice from disease of the pancreas. On the other hand, interference with the internal secretion of the pancreas is believed always to be followed by definite symptoms, *viz.*, those of glycosuria and diabetes. In acute pancreatic disease the general train of symptoms is referable to involvement of the coeliac plexus rather than to the pancreas. It consists in great pain in the upper part of the abdomen, vomiting, slow action of the heart, and collapse. The diseases of the pancreas may be considered under the headings of hæmorrhage, inflammation, tumors, cysts and calculi. The degenerative changes and atrophy require only a passing notice. The latter is common, particularly in old people, but when general over the whole organ it does not appear to cause results of importance clinically unless it be extreme, when it may cause fat to appear in the stools, and even fatty diarrhœas, particularly in children. When it affects the islands of Langerhans it may cause diabetes. Fatty changes are common. They occur under the same conditions here as in other organs, and derive their chief importance from their favouring the occurrence of hæmorrhage into the organ.

HÆMORRHAGE OF THE PANCREAS.

Etiology.—The pancreas is very prone to hæmorrhage. It arises under different conditions which are still imperfectly known. They may be grouped under two headings, viz.—(1) inflammation, (2) diseases of the vessels, such as fatty degenerations and fibrosis. The former are known as cases of *hæmorrhagic pancreatitis* and are mostly acute, the latter are chronic and may cause smaller scattered hæmorrhages or larger ones indistinguishable from those of hæmorrhagic pancreatitis. Emboli rarely lodge in the pancreatic vessels, and even when they do it is very doubtful if they cause an infarction. They may cause small hæmorrhages, but the anastomotic arterial connections are sufficient to prevent a distinct infarct. Small hæmorrhages will be replaced in time by fibrous tissue which will do little harm unless it interferes with the internal secretion of the organ. Large hæmorrhages, on the other hand, may lacerate the organ and separate large masses of it from their blood supply and thus lead to their death.

Morbid Anatomy.—Small hæmorrhages do not require any description, but the large ones cause the organ in whole or in part to be swollen and infiltrated with blood and blood clots, and sometimes to become necrosed in small or large areas. They may rupture the capsule and infiltrate the surrounding retro-peritoneal tissue or escape into the lesser omental sac.

Symptoms.—The symptoms come on suddenly in persons who have been previously in good health. They are pain, vomiting, restlessness and collapse, viz., the train of symptoms already mentioned as common to all acute pancreatic disease and believed to be due to pressure upon the coeliac plexus of nerves. The pain is severe, colicky in character, and tends to get steadily worse. It

is located in the upper part of the abdomen, but radiates in various directions. The general train of symptoms resemble those of acute intestinal obstruction. The case usually terminates fatally in two to four days.

INFLAMMATION OF THE PANCREAS.

It may be acute or chronic. Three varieties of the acute form are met with, viz., the *hæmorrhagic*, the *suppurative* and the *gangrenous*, which cannot be separated clinically from one another.

ACUTE HÆMORRHAGIC PANCREATITIS.

Etiology.—The causation of the inflammation is very obscure. There is a history of alcoholism in some, and of gall stones in other cases. The latter may act by becoming impacted in the ampulla of Vater and allowing of the passage of bile into the pancreatic duct. The experimental injection of bile or of gastric juice into the pancreatic ducts of dogs has produced hæmorrhagic pancreatitis and fat necroses. The bacillus coli and its allied germs have been found most frequently in the lesions, and it is possible that in certain states, at present unknown, of the duodenum or pancreatic juice the bacilli may acquire a heightened virulence, and, invading the pancreas, either along the duct or by the lymphatics, produce foci of inflammation or necroses and hæmorrhage which finds an easy path along the loose interlobular tissue of the gland, tearing other vessels and causing fresh bleeding as it goes, thus producing large and irregular hæmorrhages which may not only destroy or overshadow the primary foci of inflammation or necroses, but also in turn produce death of larger masses of the organ by depriving them of their blood supply. These may become gan-

grenous by the subsequent invasion of putrefactive germs from the intestine.

Morbid Anatomy.—The appearances of the pancreas and the surrounding tissues are much the same as those described under hæmorrhage. Indeed, it is often impossible to distinguish pathologically as well as clinically cases of inflammatory hæmorrhage from those of other origin. Stress must be laid upon a condition which is invariably seen in the disease, particularly as it is not limited to the pancreas, but occurs in the neighbouring omentum, mesentery and abdominal fatty tissue generally, and hence is seen early and is an important guide to the surgeon undertaking an exploratory laparotomy. It consists in the presence of a large number of foci of fat necroses. These are dead, white or yellowish-white areas of the size of a pin-head or a pea or larger, consisting of granular material, viz., fatty acids in combination with lime. They have already been described, *vide* page 21. They are most commonly seen in the hæmorrhagic and gangrenous forms of acute pancreatitis, less frequently in the suppurative. The fat-splitting ferment (steapsin) of the pancreas has been demonstrated to be present in 1 cent human and experimental cases.

Symptoms.—They are the same as those mentioned under “Hæmorrhage” in the preceding page. There is usually also constipation. The acute cases generally die in from two to four days. Some of the milder cases live for a few weeks and generally cause pyæmia. Some may recover and others may result in diabetes.

ACUTE SUPPURATIVE PANCREATITIS.

Etiology.—It probably arises much in the same way as the hæmorrhagic form, but the microbes at work produce suppuration instead of necrosis.

Morbid Anatomy.—There may be one or many abscesses or merely a purulent infiltration of the organ. Single abscesses may be large, multiple abscesses are generally small. The pus may extend to the peripancreatic tissue, the stomach, duodenum or portal vein. Foci of fat necroses are often present.

Symptoms.—They are much the same as those of the hæmorrhagic form, and may run a similarly rapid course, but some cases are chronic, lasting for a year or even more.

GANGRENOUS PANCREATITIS.

Gangrenous pancreatitis usually follows the hæmorrhagic, less frequently the suppurative. The name is used to signify that the whole organ or a considerable part of it is converted into a dry, dark or slate-coloured mass lying free in the omental cavity or attached to its surroundings by shreddy or sloughy-looking tissue, encircled by hæmorrhage or pus. The gangrenous part has been known to be discharged per rectum and the patient to recover.

Symptoms.—These are similar to those of the other forms of acute pancreatitis.

Treatment of above conditions.—The treatment of hæmorrhages and inflammations of the pancreas is chiefly surgical, but it is unfortunately the case that operation is rarely successful. The patient's strength should be maintained and symptoms treated as they arise.

CHRONIC PANCREATITIS.

Etiology.—Some of the suppurative and also of the hæmorrhagic cases may become chronic, but the chronic disease usually arises spontaneously and insidiously. In a certain number of cases there is obstruction of the duct

of Wirsung by pancreatic calculi, and occasionally by gall stones, and in some cases the fibrosis forms around a new growth. There is another group of cases in which there is an associated cirrhosis of the liver with pigmentation of the skin, intestine and liver (hæmochromatosis). In some cases no trace of a probable cause can be found.

Morbid Anatomy.—There is an increase of fibrous tissue throughout the organ, running between the lobules and among the acini. The organ as a whole may be smaller or larger than normal. In the latter case it may be palpable through the abdominal wall and thus simulate a tumor.

Symptoms.—The symptoms are very indefinite in some cases. In others they resemble those of an increasing dyspepsia. In others there are glycosuria and diabetes, as already explained, and in a few there is jaundice from the fibrous tissue in the head of the gland obstructing the common bile duct. These last cases are difficult to distinguish from cancer. The patient is generally younger, the jaundice not so severe and steadily progressive, and the course is more prolonged in the simple than in the malignant disease.

TUMORS OF THE PANCREAS.

Cancer is the most frequent and important of them. It is usually of the scirrhus type and situated in the head of the gland. It rarely causes much enlargement of the organ, and were it not for the accompanying cirrhosis it would rarely be palpable during life.

Symptoms.—*Pain* of a deep-seated and paroxysmal character is sometimes the first complaint, but the most

characteristic is *jaundice*, which is permanent and gets steadily and quickly worse, beginning as a bright yellow and going on to a dark greenish-yellow colour. The gall bladder is distended, and, when there is no ascites or marked enlargement of the liver, it is palpable. The tumor itself is said to be palpable in more than a quarter of all cases. The stools are clay-coloured but seldom show fat. Diabetes is rare. Emaciation appears early and rapidly develops. Death follows in a few weeks to a few months, rarely as late as a year, after the jaundice shows itself.

Treatment.—An attempt can be made to supply pancreatic secretion where the tumor interferes with the duct of *Wirsung*, but, as a rule, beyond treating symptoms and supporting strength nothing further can be done.

CYSTS OF THE PANCREAS.

The most interesting and important clinically are the large peritoneal cysts situated usually in the lesser omental sac, which contain a dirty looking fluid, amounting sometimes to several pints, often brownish or reddish in colour, and generally mixed with blood. They have been said to follow trauma, but they do not all do so. Some may be the result of a mild hæmorrhagic pancreatitis from which the patient recovered. In others no explanation of their origin can be given. The fluid generally contains the amylopsin ferment of the pancreatic juice in distinct amount, and sometimes also the trypsin ferment in small amount. Cysts of another kind altogether may be found within the substance of the organ. They are nearly always retention cysts from pancreatic calculi, much more rarely from gall stones, blocking the duct of *Wirsung*, or new

growths in the head of the organ or bile duct or duodenum acting in the same way. The duct of Wirsung may in consequence be uniformly dilated, often to the size of a finger, or be distended into numerous lateral dilatations or cysts. They frequently contain pancreatic calculi, and their fluid contents give for a time the reactions of the pancreatic juice, and later those of pus, mucus or blood. Another form of cyst much more rarely met with is the congenital, associated with similar cyst formation in the kidney and liver. The cysts in this case are usually small but occasionally the whole organ is converted into cysts of some size.

Treatment.—The cyst, when of sufficient size to be diagnosed, should be tapped, or if very large it may be drained after stitching to the abdominal wound.

CALCULI OF THE PANCREAS.

They are not common, but occur much more frequently in males than in females, and thus contrast strongly with gall stones. They are probably due to inflammation of the ducts set up by germs coming from the intestine. They are mostly multiple, smooth or rough, round, oval or irregular, white or grey in colour, soft in consistence, and vary in size from mere specks to the bulk of a walnut. They are mainly composed of carbonate of lime. They may cause dilatation of the ducts, cirrhosis of the gland, suppuration, or acute inflammation. They have rarely been found associated with cancer, so that they can hardly be said to favour its occurrence. This, again, contrasts strongly with gall stones.

Symptoms.—There may be no symptoms or there may be paroxysmal pain, jaundice, glycosuria or fatty diarrhoea.

Treatment.—If diagnosed they should be removed as soon as possible.

X.—DISEASES OF THE PERITONEUM.

The diseases of the peritoneum may be grouped under the headings of circulatory disturbances, inflammations, new growths and cysts.

CIRCULATORY DISTURBANCES.

Hyperæmias and dropsy or ascites are the circulatory disturbances met with in the peritoneum. Hyperæmia may be active or passive. The former is met with—(1) as a local condition in many gastro-intestinal affections, *e.g.*, hernia, (2) as a general condition in consequence of sudden diminution of the intra-abdominal pressure, as in removal of a tumor or ascitic fluid. The latter is part of a general venous congestion in chronic cardiac and lung disease and part of a local abdominal congestion in obstruction to the portal circulation, either within or without the liver. *Ascites* is the result of such long-standing venous congestions, though it is not entirely caused by them. In most cases, if not in all, a toxic influence is probably also at work rendering the vessels of the peritoneum more permeable. Tumors of the abdomen, particularly of the ovaries, must be mentioned as causing considerable ascites, and the obstruction to the portal circulation cannot in their case be the only cause. The ascitic fluid varies in amount. When large it causes the navel to become bulging and prominent. Its character also varies, but it is usually clear and watery, though it may contain blood pigment, bile, or fibrin flakes. It is generally free in the

peritoneal cavity, and changes its position, in obedience to the law of gravity, with changes in the posture of the patient. It may be encysted, particularly in children, in whom it often collects between the layers of the great omentum (hydrops omenti). In some cases the fluid is milky from an admixture with chyle following rupture of the thoracic duct or other abdominal lymph channels (chylous ascites).

Symptoms of Ascites.—A gradual uniform enlargement of the abdomen, which causes it to become rounded, and leads to prominence and projection outwards of the navel, is the most characteristic feature. It is associated with an upward displacement of the diaphragm, which may thus press upon the heart and lungs and hamper their action. On palpating with the left hand applied to the right side of the abdomen and a smart tap given with one of the fingers of the right hand to the left side of the abdomen, a fluid wave is felt to strike the fingers of the left hand. Comparatively small amounts of fluid may be detected in this way. In fat people a wave may be transmitted through the abdominal wall and give a false impression to the observer. This may be avoided by an assistant placing the edge of his hand or the edge of a book or piece of cardboard lightly in the middle line of the abdomen. Percussion gives a dull note over the fluid and a tympanitic one elsewhere. In the dorsal position the intestines float upwards upon the fluid, hence the flanks are dull and the front of the abdomen is tympanitic. The fluid must reach between two and three pints (probably) before it will give rise to dulness on percussion. The dull area will change with the posture of the patient, always occupying the most dependent parts, except when the fluid is very cysted, when it will change its position either slightly or not at all. The knee-elbow position is a good one for bringing out small amounts of fluid.

Treatment.—It is needless to say that the line of treatment must depend on the cause of the condition. In most cases of ascites the fluid may be reduced by free purgation and diuresis, and also by curtailing the amount of fluid imbibed by the patient and eliminating salt from the dietary. There is some difference of opinion as to the wisdom of early aspiration, because the peritoneal sac re-fills with fluid at a rapid rate, and the consequent drain on the patient's lymph is very great. Southey's tubes should be used, the site of operation being in the mid-abdominal line, between the umbilicus and the symphysis pubis. Before inserting the needle, ascertain that the bladder is empty, and remember during the operation to keep up pressure by an abdominal bandage whilst the fluid is gradually withdrawn. Soon after aspiration is completed resume the use of purgatives and diuretics. Diuretin in 10 to 15 grain doses, and the *spiritus etheris nitrosi* in drachm doses, will be found excellent diuretics. Should the ascites be of malignant origin tapping may have to be repeated at frequent intervals, while in tubercular cases the performance of abdominal section and washing out the peritoneal sac has sometimes aborted the tubercle.

INFLAMMATIONS—PERITONITIS.

Etiology.—In treating of the inflammations of other serous membranes, *e.g.*, pericarditis, pleurisy, it was pointed out that it was essential, both on etiological and clinical grounds, to draw a distinction between primary and secondary inflammations. It is equally important to do so in the case of the peritoneum, though a primary peritonitis is of doubtful occurrence. Primary, compared with secondary pleurisy is rare, though it occurs fairly frequently. Primary pericarditis is rarer, while primary peritonitis is

so rare that its occurrence at all is open to doubt. The cases of peritonitis which have been held to be primary have occurred after a chill or apparently spontaneously. The former are believed to be rheumatic in nature, and other evidences of rheumatism can generally, if not always, be found, though they are insignificant beside the peritonitis. The latter, the so-called spontaneous form, can always be explained by some general infection or pre-existing disease. Secondary peritonitis, on the other hand, is of frequent occurrence. It may be grouped under three divisions—(1) from without, (2) from some abdominal organ, (3) from disease elsewhere in the body. The first division is comparatively uncommon. It is exemplified by perforating wounds of the abdominal wall or operations upon the abdominal cavity with faulty asepsis. The second division is the most important. It occurs in perforations of the gastro-intestinal tract, of the gall bladder, of abscess of the liver, spleen, pancreas, kidney, retro-peritoneal tissues or pelvic organs, *e.g.*, ovary, tubes, or as an extension from any of these organs when inflamed, suppurative, necrotic or the seat of malignant disease without actual rupture. Appendicitis and salpingitis must be specially emphasised as they are the most frequent of all the causes of peritonitis. It must be noted, too, that all perforations of either the abdominal wall or the intestine are not followed by peritonitis. The healthy peritoneum has great microbic powers and can protect itself against doses of bacteria which are not too large. The dose which can be taken without setting up peritonitis varies with the individual and with the virulence of the germ, but some people can withstand considerable doses. In recent warfare there have been numerous instances of perforations by bullets of the abdominal wall and of the gut, which healed kindly without the onset of peritonitis. It is probable that the high velocity of the bullet rendered the abdominal wound practically aseptic and the small

aperture, both in the abdomen and the gut, favoured the rapid sealing up of the apertures and prevented extensive bacterial invasion of the peritoneum. The third division mentioned above is represented by general diseases such as pneumonia, tubercle and pyæmia, and by local diseases which lead to general lowering of the vital powers, such as Bright's disease, in which peritonitis is a common terminal event. The bacteriology of peritonitis is thus of less importance than it would be if it were a common primary lesion. Its importance is, nevertheless, sufficient, both on practical and prognostic grounds, to entitle it to careful consideration. In many of the diseases mentioned above a mixture of germs may be present, and hence the consequent peritonitis may show the same mixture or only some one or more of those present in the primary morbid state. The ordinary non-specific germs of acute inflammation and suppuration have been found most frequently, viz., the pyogenic cocci, streptococci and staphylococci, and the bacillus coli, and it does not seem to be necessary to calculate which of them occurs oftenest. The diplococcus lanceolatus (Frænkel) is rarer, and the bacillus pyocyaneus still rarer. Among the specific germs, the gonococcus, the tubercle, typhoid and influenza bacillus have been met with, the first two comparatively frequently, and the last two more rarely. The gonococcus is particularly interesting as the peritonitis caused by it nearly always spreads from salpingitis and may be met with in children. It is rarely general, but whether general or local it runs a mild course and never requires operative interference.

Morbid Anatomy.—The morbid changes are similar to those seen in other serous membranes, *e.g.*, the pericardium, pleura and meninges. They come on with great rapidity, even in a few hours, in rapid cases proving fatal within about twenty-four to thirty-six hours. There may be merely injection of the vessels of the serous surfaces with

so little exudation as to be almost invisible to the naked eye. In cases which live long enough there is considerable exudate of a serous, sero-fibrinous, fibrinous, purulent or hæmorrhagic character. The serous exudate tends to accumulate behind the coils of bowel and in the pelvis, and may be of considerable amount. The fibrinous exudate may cover the intestinal coils with white masses or, more commonly, glue the coils together or form flakes floating in the fluid. The coils are easily separated from one another, but the bowel wall is so friable that the serous coat is often torn away in the separating. The purulent exudate (and most cases tend to become rapidly purulent) is either a thin greenish-yellow or a creamy-white fluid. It usually collects in "pockets" among the intestinal coils. The hæmorrhagic exudate is seen in cancerous, tubercular and wound peritonitis. In most cases the intestines are distended with gas from paresis or paralysis of their muscular coats. The peritoneal cavity may also contain food from the stomach, fæces from the intestine, bile from the gall bladder, or urine from the urinary bladder in perforation of these viscera. This description applies to acute general peritonitis, though the inflammation is often greatest at the place where infection of the peritoneal cavity took place. In many cases it may be more or less limited to these areas, forming a local peritonitis. The most important of these local forms require separate consideration.

Symptoms.—The symptoms arise either from the morbid changes in the abdomen or from the toxæmia. The most important of the former are pain and tenderness in the abdomen, retching and vomiting, contracted or distended abdomen, effusion into the peritoneal cavity and constipation, and of the latter; fever, quick pulse, leucocytosis, the "hippocratic facies," and collapse. They are generally all present, though varying in degree in typical cases in,

those arising suddenly in fairly vigorous cases from perforation of an abdominal viscus, from pelvic inflammation, or from puerperal infection. Many of them may be absent in young or old people or in those debilitated by serious disease, *e.g.*, typhoid fever, cancer or tubercle. Variations in the symptoms are best understood when those due to the action of the irritant are distinguished from those caused by the reaction of the system. The *pain* is due to both factors. It is caused by the escape of the infectious visceral contents (action) and the consequent congestion (reaction) stimulating the nerve endings over a large surface. Therefore it is usually sudden in onset and tends to increase in severity. If the sensorium is dulled by previous poisoning, it may be slightly or not at all appreciated. It will be increased by anything which causes the inflamed surfaces to be rubbed or pressed together, such as muscular movements, deep breathing, coughing, sneezing, vomiting, borborygmi, &c. Hence the patient assumes the dorsal position with the legs drawn up. The abdominal muscles are firmly contracted and flattened so as to be as rigid and motionless as possible. The breathing is shallow, rapid and actively thoracic. Coughing, &c., is dreaded and suppressed as much as possible. There is generally also great tenderness, so that even the weight of the bedclothes cannot be tolerated. As the pain increases, the patient gets restless, cannot keep still notwithstanding the aggravation which the movement causes, and frequently cries out in agony. The pain and the dread cause the face to become drawn, anxious-looking, and covered with sweat. Occasionally the pain is slight when the patient is able to maintain a position of perfect rest. Sudden relief or cessation of the pain suggests the formation of pus. The *retching and vomiting* are due partly to reflex stimulation from the peritoneum and partly to the action of the toxins upon the vomiting centre in the medulla. They usually come

on early and last till the end. After the stomach contents are voided, the vomit consists chiefly of fluid, at first yellow and later green from bile staining. Towards the end it occasionally becomes brownish, with a fæcal odour, and may be drawn into the lungs and cause death. The *abdomen*, as already mentioned, is at first retracted and flat. It may feel as rigid and motionless as a board. It may remain so to the end, particularly in perforation cases, but it usually gradually gives way to distension, which may steadily advance until the abdomen assumes a rounded shape. It is still motionless. It is highly tympanitic. The diaphragm and apex beat are pushed upwards and the liver and splenic dulness partially or entirely disappear. This abdominal distension is due to the collection of flatus in the bowel following the relaxation and paresis of its muscular wall. It is part of the local reactionary inflammation. The *effusion* into the peritoneal cavity is of the same nature. It varies in amount, but is present in most cases after about thirty-six to forty-eight hours from the onset of the disease. It may give rise to dulness and sometimes fluctuation in the flanks. The *constipation*.—Diarrhoea is frequently present at an early stage. It usually gives way to constipation, but may persist, particularly in puerperal cases. Occasionally the bowels act naturally throughout. The only other alimentary symptoms which need be mentioned are dry tongue and lips, sordes on the teeth, and great thirst. These local symptoms are nearly altogether reactive to the infection of the peritoneum, and consequently they take time to develop. They are best seen in cases which do not prove fatal until four, five or more days have passed. The more rapid the case the slighter they are. Death in the rapidly-fatal cases is ascribed to collapse, formerly thought to be due to shock, but now believed to be chiefly caused by toxæmia. The general symptoms will vary in the same way with the rapidity of the disease.

In the most severe and rapid cases, the symptoms are due to the direct action of the poison upon the nerve centres, hence in them we see only great prostration and depression, the pinched face, dry or covered with clammy sweat, the sunken eyes, the weak heart, the cold extremities and general collapse, but little or no fever and no leucocytosis. The two last symptoms are due to the reaction of the system to the poison, and hence are best seen in cases which are not overpowered quickly by the poison, such as those running a less rapid course, particularly in fairly robust patients. A few particulars of these symptoms may be given. The temperature may follow rapidly upon a rigor, but rigors are more common in the course of a case than at its onset, and they then usually indicate the onset of suppuration, when the fever also generally drops. The temperature usually rises quickly to about 103°F . or higher, and mostly runs an irregular course. For reasons already given, when the temperature remains subnormal for two days or more the outlook is very grave. The *pulse* gives valuable indications of the progress of the case. It early becomes rapid, about 110. It tends to increase, and may in a day or two reach 150 or even 200, and is usually most rapid in very severe cases. It is small and hard and is described as possessing a "wiry" character. The *leucocytosis* may quickly reach a high figure, as much as 100,000 per cmm. Its appearance in a case of typhoid fever (in which there is no leucocytosis) should thus suggest the onset of perforative peritonitis. The *urine* is scanty, high-coloured, and contains much indican and many urates. Micturition is generally frequent and painful. The *signs of collapse* have already been referred to. The pinched face, the sunken eyes, the anxious expression, the slightly dusky or livid hue which are so often seen in cholera, as well as in this disease towards the end, may be absent, and the face may be only flushed and perspiring. The duration of the

disease varies from about thirty-six hours to seven days or more.

Diagnosis.—Great care must always be exercised in coming to a decision, for, while a correct diagnosis may be made with fair certainty in cases where the typical abdominal and general symptoms are present, particularly when a history of gastric ulcer, appendicitis or some other disease can be obtained, it is to be remembered that acute general peritonitis varies in its clinical manifestations more than most other diseases. Intestinal obstruction, acute pancreatitis, acute entero-colitis, embolism of superior mesenteric artery, or ruptured tubal pregnancy are most likely to be mistaken for it. The later in the disease the patient comes under observation, the more difficult does the examination become. A pelvic examination in female cases will frequently give important signs.

Prognosis.—It is nearly always fatal, particularly in children and old people. Gonorrhœal peritonitis forms, as already mentioned, a prominent exception, for, if uncomplicated, it always runs a favourable course and recovers. Pneumococcal peritonitis is also fairly favourable, but nearly all other forms, if general, kill within four or five days on the average. Death may be sudden from cardiac failure.

Treatment.—The patient should be kept absolutely at rest in bed and the abdomen covered with a cage so as to keep off the weight of the clothes. The question of operation should not be obscured by the administration of morphia, because soothing the pain may mean the sacrifice of the patient owing to delay in operating. In hopeless cases, on the other hand, or where operation is for some reason impossible, give morphia freely. Locally apply either heat or ice, certainly the latter is preferable in most

cases. Relieve abdominal distension by introducing a long rectal tube and, if that fails, puncture one or more coils of the intestine through the abdominal wall with a fine aspirating needle. Enemata sometimes afford relief, and rectal alimentation is the only possible means of keeping up the patient's strength. For the vomiting, the stomach should be washed out, and small pieces of ice administered from time to time. Hiccough is usually relieved by sedatives and sometimes by the application of a mustard leaf over the stomach.

ACUTE LOCAL PERITONITIS.

The inflammation may be confined to a part of the peritoneum, in these cases it is prevented by limiting adhesions from spreading to the rest of the peritoneal cavity. There are three important forms distinguished by their position, viz., upper or subphrenic, lower or pelvic, and right-sided or appendicular.

SUBPHRENIC OR SUBDIAPHRAGMATIC ABSCESS.

Etiology.—A gastric ulcer which has leaked or perforated is the commonest cause, but trauma and severe or suppurative inflammations of other organs, viz., the pancreas, liver, gall bladder, spleen, duodenum, kidney and vermiform appendix also cause it. The abscess will, with occasional exceptions, lie to the left side of the falciform ligament of the liver if the originating lesion is to the left of the pylorus, and to its right side if it lie to the right. It has been thought to follow upon pneumonia, but its origin within the thorax is doubtful.

Morbid Anatomy.—The size of the abscess varies greatly, and its boundaries vary in a corresponding manner. In

left-sided abscesses two boundaries are constant, viz., the diaphragm above and the anterior abdominal wall in front. The left and right boundaries vary. When small they are bounded to the right by adhesions between some part of the anterior surface of the left lobe of the liver and the anterior abdominal wall, and to the left by adhesions between some part of the anterior stomach wall and the anterior abdominal wall. When larger they may extend to the falciform ligament of the liver and to the lower ribs and spleen respectively. It is bounded posteriorly by part of the anterior wall of the stomach and higher up by the thickened anterior wall of the lesser omental sac or by general adhesions of the posterior peritoneum covering this part of the posterior abdominal wall. The contents may be purulent, seropurulent, and contain food, detritus or air. When air or gas is present it has been named "subphrenic pyo-pneumothorax." The gas is usually quickly absorbed, and if the originating rupture in the viscus is closed by lymph, as often happens, it is not renewed unless in the rare cases where gas-forming bacteria are present and active. Occasionally the fluid is restricted to the sac of the lesser omentum. This is particularly liable to occur in pancreatic cases when the fluid is frequently hæmorrhagic. In these cases the stomach lies in front of the swelling, the pancreas behind it, and the colon below it. Right-sided abscesses have also the same two constant boundaries, viz., the diaphragm above and the anterior abdominal wall in front. The posterior boundary is the anterior and upper surface of the liver, so that the organ is pushed downwards proportionately to the size of the abscess. Subphrenic abscesses, whether right or left sided, show a remarkable tendency to perforate the diaphragm and invade the pleura and lung, less commonly the pericardium. Pleurisy, pneumonia or gangrene of the lung may follow after the subphrenic abscess has been in existence for only a short time. Rupture into the general

peritoneal cavity is not to be feared, but it may occur into the stomach or colon, though it is rare.

Symptoms.—The symptoms are variable, but many cases begin with severe pain in the upper part of the abdomen, followed by vomiting, the appearance of a swelling, and embarrassed respiration. The symptoms due to toxæmia gradually follow, viz., rigors, fever, rapid pulse, anæmia, and leucocytosis. The picture is somewhat like acute general peritonitis on a small scale. The existence of the abscess may be demonstrated by the Röntgen rays. Symptoms of thoracic implication soon appear and the diagnosis may be obvious, but it remains doubtful in many cases. The early implication of the thorax makes the prognosis graver than it otherwise would be. Early operation is the best hope, but the outlook is not very bright. Recovery occurred in about 20 per cent. of the cases which have been put on record.

Treatment.—The treatment has just been indicated; it is only necessary to add that symptoms as they arise may claim attention.

PELVIC PERITONITIS.

Reference has already been made to the frequency and comparative mildness of acute gonorrhœal peritonitis. It spreads from the Fallopian tubes. They are the most common source of all forms of pelvic peritonitis. The fimbriæ become adherent to the ovary and the tubes greatly dilated. Puerperal infection and tubercle are other causes of pelvic peritonitis.

Treatment.—The treatment of this condition belongs to the domain of the specialist, but douching and counter-irritation over the ovarian regions may be mentioned as the methods usually adopted.

APPENDICULAR PERITONITIS.

This has already been sufficiently considered under appendicitis.

CHRONIC PERITONITIS.

Etiology.—It may follow the acute form or arise independently. In the latter case it is often tubercular or cancerous, while in other cases no cause can be discovered.

Morbid Anatomy.—There may be merely one or more bands of fibrous tissue, or there may be general thickenings involving a great part or a whole of the peritoneal surfaces. The pelvic region deserves special mention, as it is a very common site of chronic inflammation, resulting usually in adhesions and displacements. When the peritonitis is general it forms thickened, white, opaque surfaces in the bowels, omentum and parietal peritoneum. In the tubercular and cancerous forms these thickenings are accompanied by extensive adhesions between the various thickened surfaces, so that the coils of bowel are matted together and cannot be separated without tearing them, while in another form, sometimes called the proliferative, there are few adhesions. This form produces a great thickening of the capsule of the liver, a general perihepatitis, in which the liver, often itself cirrhotic, is surrounded by a dense white jacket, one quarter inch or more in thickness, which easily peels off. A similar perisplenitis is seen. These changes are not infrequent in alcoholics. In all forms of chronic peritonitis the great omentum tends to be retracted and thickened into a transverse mass in the upper part of the abdomen.

Symptoms.—The most characteristic and important symptom is *ascites*. Frequent tapping is required, as the fluid tends to re-accumulate rapidly. Anæmia and wasting supervene. Constipation is frequent at first, followed by diarrhoea. The patient usually dies of exhaustion.

Treatment.—Tapping may require to be carried out at regular intervals, although an attempt should be made by purgation, diuresis, dry diet, and diminishing the saline constituents of the food to reduce the rate of accumulation of the fluid. In certain cases counter-irritation with iodine is advantageous.

NEW GROWTHS IN THE PERITONEUM.

Malignant disease is the most important. It is very rarely primary. In this case it is an endothelioma, though a primary colloid cancer has been described on what must be looked upon as doubtful evidence. The endothelioma forms a single or multiple growth, varying in size; the colloid cancer, an enormous growth which fills the whole peritoneal cavity and coats every organ as if a fluid had been poured into the peritoneal cavity and had subsequently coagulated. Secondary cancer spreads most frequently from the stomach or the pelvic organs. It forms numerous nodules of varying size, often umbilicated, in the parietal peritoneum and also in the mesentery and walls of the bowels. Chronic peritonitis results, matting the organs together and shrinking up the omentum into a mass lying transversely across the upper part of the abdomen. There is considerable ascites, often collected into pockets.

Symptoms.—In most cases the symptoms of the primary lesion have disclosed the nature of the case. If they are

absent a diagnosis is difficult. Ascites, frequently hæmorrhagic, is the most common symptom. Thickenings, &c., may be felt through the anterior abdominal wall.

Treatment.—Ascitic effusions should be tapped when necessary, symptoms must be treated as they occur. Pain demands the free use of opium.

PERITONEAL CYSTS.

Etiology.—Some arise from injury, others from the pancreas, and others are of unknown origin. They are situated most commonly in the upper part of the abdomen in the middle line. They vary in size and contain a brown or reddish-brown fluid, in which red and white blood corpuscles, blood crystals, and sometimes also large endothelial cells are to be found. The fluid may contain amylopsin and trypsin.

Symptoms.—Colicky pain and vomiting are present for a time and then a tumor appears.

Treatment.—The treatment is surgical.

Section 7.

DISEASES OF THE KIDNEY.

MALFORMATIONS AND MALPOSITIONS.

1. A considerable number of malformations of the kidney are described, but they are not of much clinical interest. The most important of them is that in which only one kidney can be detected during life by palpation and percussion. This may be due to one kidney being undeveloped, to its atrophy during or after development, or to both kidneys being present but fused into one mass. The commonest form of the fused kidney is the horse-shoe form, where a bridge of kidney tissue unites the lower ends of the two fairly well-formed organs. When only one kidney is present, it undergoes compensatory hypertrophy.

2. The anomalies in position are also several in number, downward displacements towards or into the pelvis being the commonest. A minor form is the so-called *movable kidney*, in which the organ is more or less freely movable, due either to a looseness of its surrounding tissues, viz., the fatty tissues which form its bed, and the peritoneum, which is reflected over its anterior surface, or to its being more or less fully surrounded by peritoneum and possessing a mesonephron. The degree of mobility is greatest in the latter case, sometimes called also "floating kidney," which is much rarer than the other.

MOVABLE KIDNEY.

Etiology.—The greatest degrees of mobility are probably congenital, while the minor degrees are acquired. Whether congenital or acquired, it tends to become exaggerated as time goes on, and the causes which operate are laxity of the abdominal walls, of the peritoneal folds, of the perirenal fat, and depression by the liver or dragging down by tumors. The mobility of the kidney is combined in some cases with displacement downwards of the other abdominal viscera and with neurasthenia, a condition known as “enteroptosis.” Movable kidney is more common (about seven times) in women than men, on the right than on the left side, and when present on both sides is more marked on the right. It may lead in some cases to kinking of the ureter with consequent hydronephrosis, or of the renal vein with consequent passive congestion.

Symptoms.—In most cases of slight mobility (where the lower part of the organ merely is palpable) there are no symptoms. In a small proportion of cases, particularly those of considerable mobility, the symptoms are severe. In the great majority of all cases the symptoms, if any, are comparatively slight. Pain in the abdomen or loin is the most frequent of them. It may be either intermittent or constant, with exacerbations at times. Bi-manual palpation of the kidney (the left hand being placed posteriorly in the lumbar region and the right anteriorly in the hypochondriac region) may elicit tenderness or a dull pain. During paroxysms of severe pain there may be nausea and vomiting, fever and collapse. Intermittent hydronephrosis or hæmaturia may arise from kinking of the ureter or of the renal vein respectively, in which cases the pain may be very severe and simulate renal colic. In one type of case there is dilatation of stomach with dyspeptic symptoms, probably of mechanical origin. Loss of flesh and nervous depression

gradually follows. In another type of case there is no dilatation of stomach or dyspepsia but marked nervous symptoms, *e.g.*, headache, drowsiness and nervous depression, probably from renal inadequacy. Vomiting is not common, but loss of flesh comes on and the nervous depression may become severe and even end in mania.

Treatment.—A kidney which is movable, and especially one which is floating, requires the use of a broad abdominal bandage, sometimes with a pad so applied as to keep the kidney in position. In cases in which the floating kidney causes excessive pain, abdominal section should be performed and the kidney stitched to its proper attachment (Nephrorraphy). Recent modifications in the surgical procedure have resulted in permanent cure.

For paroxysms of pain, rest in bed, fomentations and linseed poultices should be applied. Sometimes the pain is so excessive that opium or other sedative is necessary.

DEGENERATIONS.

The kidney is liable to granular and fatty as well as to waxy degeneration, but the two former do not *per se*, *i.e.*, apart from some general disorder or some inflammation of the kidney, give rise to such definite changes as to constitute distinct kidney disease recognisable clinically, whereas the latter, *viz.*, waxy degeneration, may, apart from its causal condition, produce such definite morbid changes in the kidney as to focus attention upon that organ during life.

WAXY DEGENERATION OF THE KIDNEY.

Etiology.—This has already been considered under the heading of general waxy disease. Tubercle, syphilis and

prolonged suppuration are the main causes, and it is to be remembered that their preceding existence may have to be searched for with great care. There is no difficulty in many cases in finding evidence of the one or the other, but in other cases the evidence is slight. Some deformity, *e.g.*, a defective joint, may point to tubercle, or a tropical complexion may suggest dysentery or liver abscess. For instance, prolonged suppuration from the bowel may have been present for a long period without being ascertainable from the patient, or at any rate without leaving any external sign.

Morbid Anatomy.—In uncomplicated cases, *i.e.*, those in which no other morbid change in the kidney is combined with the waxy disease, the organ is usually somewhat increased in size and paler than normal. When this increase in size is considerable it constitutes one of the varieties of “large white kidney.” This term has no definite pathological significance, but merely indicates that the kidney is markedly larger and paler than the normal organ. In the earlier stages of the disease there is no increase in size, nor is there any pallor or other morbid change distinguishable macroscopically, except it may be a prominence of the glomeruli, until the iodine reaction brings out the characteristic brown spots and lines which indicate that waxy degeneration is present in the glomeruli and in the vessels. As the disease advances, the size and pallor of the organ increase, until the whole cortex becomes opaque white, with yellow spots and streaks (the result of co-incident fatty degeneration), while the bases of the pyramids are congested. The capsule is not thickened or adherent. The surface of the kidney remains smooth, but the glomeruli become more prominent, and iodine gives a more extensive and diffuse waxy reaction, both in the cortex and in the pyramids. The causes which induce waxy degeneration are liable to provoke ordinary inflam-

mation as well, hence kidneys affected with waxy disease frequently suffer also from either parenchymatous or interstitial inflammation, or both combined. The former increases the pallor and fatty degeneration of the organ, while the latter causes thickening and adhesion of its capsule with alterations in the size of kidney itself, varying according to the degree of the complicating changes. Thus waxy diseases may be associated with the chronic parenchymatous or with the large or small granular kidney. The microscope shows that the waxy change is extensively distributed throughout the kidney structures, the following being the order in which they are affected, approximately so at any rate, viz., the walls of the afferent arterioles and of the glomerular capillaries, of the straight vessels and their capillaries in the pyramids, of the efferent arterioles and their intertubular capillaries, of the capsular epithelium of Bowman and of the glomeruli, of the larger vessels, &c., the epithelium of the convoluted tubules being one of the latest structures to be affected.

Symptoms.—It is the less necessary to distinguish between uncomplicated and complicated cases of waxy disease seeing that the general symptoms of both waxy disease and chronic nephritis are very much alike. Polyuria is the most marked and frequent symptom of early waxy disease. It is impossible to say how early it begins, but in all probability it does not do so until the disease has been in existence for a considerable time. The increase in amount may be slight or may be three or four times the normal. It generally tends to get greater for a time and to be free or nearly so from albumin and casts. Later on the amount of urine gradually diminishes, and that of the albumin correspondingly increases, until they present very much the proportions of ordinary chronic Bright's disease. Casts, on the other hand, are of comparatively early and frequent occurrence, being chiefly of the

hyaline variety. They become more frequent and varied (fatty, granular and epithelial) in the later stages. Some authorities hold, perhaps on insufficient grounds, that true waxy casts giving some or all of the staining reactions of waxy material are occasionally, though rarely, met with. Diarrhoea is another frequent and early symptom. It is generally attributed to the intestine having become affected by the waxy disease, but recurrent enteritis, to which the waxy intestine is prone, has a good deal to do with it. Waxy disease is generally present also in the liver and the spleen. Dropsy appears in over two-thirds of all cases, and mostly only in the later stages, when the diuresis and diarrhoea have subsided. Thus the dropsy and albumin tend to increase as the diuresis and diarrhoea diminish, and *vice versa*. Anæmia is present early, gradually increases and becomes pronounced. The further course of the disease runs much along the lines of ordinary chronic Bright, except that there is usually little or no arterial fibrosis or cardiac hypertrophy. Apoplexy is thus relatively infrequent, but the ordinary inflammatory complications of Bright's disease are common, such as pericarditis, pleurisy, pneumonia, peritonitis, &c.

Diagnosis.—Polyuria and diarrhoea in a case with a history of tubercle, syphilis, or prolonged suppuration (abscess of liver, dysentery, &c.) are the most reliable guides, but some cases run a more or less latent course throughout.

Treatment.—The treatment is unsatisfactory. If the case is a syphilitic one give potassium iodide; if the result of bone disease treat the cause where possible. Sooner or later a cardiac tonic will be required. Tap ascitic effusion and hydrothorax when necessary, and give a salt-free dietary a fair trial. Treat other symptoms as they occur; dyspepsia, vomiting, and diarrhoea will probably call for special attention.

*DISTURBANCES OF THE RENAL
CIRCULATION.*

ACTIVE CONGESTION.

Active congestion is so similar to the early changes of nephritis that it does not require separate description.

PASSIVE CONGESTION.

Etiology.—Chronic cardiac and chronic lung diseases produce varying degrees of renal congestion, and so do all diseases of the mediastinum and abdomen which are attended with obstruction to the venous return through the inferior vena cava, but the most extreme degrees of the condition occur in thrombosis of the renal vein, a comparatively rare consequence of renal cancer, pyelitis, granular kidney, pelvic thrombosis, or of some general infection.

Morbid Anatomy.—The kidneys are swollen and dark red in colour, the *venæ stellatæ* and the glomeruli are enlarged and prominent and there may be many small hæmorrhages, both in cortex and medulla.

Symptoms.—A small quantity of highly-coloured urine of high specific gravity and rich in urates is voided. Albumin is present, generally in fair quantity, though not so large as usually occurs in nephritis. Blood, hyaline and granular casts are frequent. When dropsy is present it is due to the cardiac and not to the renal changes.

Treatment.—Relieve backward pressure, treating the cardiac and pulmonary causal conditions by cardiac tonics, diuretics and purgatives.

INFARCTIONS.

The subject of infarction in general has already been described.

Etiology.—Infarction of the kidney is caused by the obstruction of a branch of the renal artery.

Morbid Anatomy.—A necrotic wedge-shaped area is produced, of a red or white colour, according as it occurs in the medulla or cortex. Later on it becomes encapsulated and undergoes absorptive changes, resulting in the production of a cyst or cicatrix.

Symptoms.—In many cases the symptoms are so slight and indefinite as to pass unnoticed, in others they resemble those of venous congestion or acute Bright's disease. Albumin tends to appear suddenly and to disappear as suddenly. Its amount is not usually large. Blood usually appears and disappears with similar suddenness, but its amount is often large. Occasionally the occurrence of a passing hæmaturia and albuminuria may enable a diagnosis to be made, particularly when some condition, in which emboli are probable, is present, such as ulcerative endocarditis.

Treatment.—When pain is present apply fomentations or linseed poultices. It is rarely possible to do anything further.

*DISTURBANCES OF THE RENAL
SECRETION.*

The amount and composition of the urine remain fairly constant in health. Structural or even functional

disorders, either of the kidney itself or of some other organ, may lead to changes either in the amount and proportion of the normal constituents of the urine or to the appearance in it of abnormal substances. Among the more important of the former are alterations in the amount of water, of certain nitrogenous extractives, such as urea, uric acid and kreatinin, of urinary salts and pigments, and among the more important of the latter are albumin, blood, pus, sugar, fat, acetone and abnormal pigments. A short summary of the conditions under which each of these changes occurs is useful.

THE NORMAL CONSTITUENTS.

Water.—The quantity of water voided in the urine in health averages from 40 to 50 oz. In health it may occasionally fall to 20 or rise to 80 oz. Such variations depend upon both the quantity of fluid ingested and upon the amount of fluid eliminated by other channels, viz., the skin, lungs and intestine. In disease the variations are much greater. An increase in amount occurs both in certain kidney diseases and in certain structural and functional changes in other parts of the body. Among the former, the most important are waxy degeneration (the early stages of the uncomplicated forms), granular contracted kidney and chronic parenchymatous or chronic diffuse interstitial nephritis when dropsy is absent or when it is subsiding; while among the latter are diabetes mellitus, diabetes insipidus and certain cardio-vascular conditions. A diminution in amount is of still more frequent occurrence, both in renal and other diseases. It is seen in the acute and in most of the chronic forms of nephritis, in venous congestion, and all other diseases which lessen the flow of blood through the kidneys, e.g., chronic cardiac and pulmonary diseases, in dropsy, whether of cardiac, renal or hepatic origin, in profuse diarrhoeas (cholera), and in

all febrile disorders. It may amount to complete suppression or *anuria*, of which two varieties are described, viz., the obstructive and non-obstructive.

Anuria.—The obstructive form occurs when both ureters are blocked, *e.g.*, by calculi or by cancer of the bladder or uterus involving both ureters. The urine continues to be secreted for a time, though none is passed, since it collects in the renal pelvis, causing an acute hydro-nephrosis; later the secretion ceases. The non-obstructive form is met with, not only in serious kidney disease, such as intense acute nephritis and in the later stages of chronic Bright's or of cystic disease, but also in diseases of other parts of the body, without the kidneys themselves being affected, the result probably of some reflex nervous action. Thus suppression has followed upon an exploratory incision into the kidney, upon some general surgical operation, or even upon the passing of a catheter. It may occur also in the course of some general infection or fever, such as diphtheria or yellow fever, or in the collapse stage of cholera, or in cases of poisoning by phosphorus, lead or turpentine. The symptoms of the non-obstructive form, and of some of the obstructive ones, are the same as those of ordinary acute and chronic uræmia, but those of certain of the cases, probably the majority, of the obstructive form differ considerably therefrom. They are grouped under the term "latent uræmia," owing to the disturbances being comparatively slight. They consist of headache, vomiting, and occasionally convulsions. Consciousness is retained.

The specific gravity.—The average specific gravity of the urine is about 1020. Persistent low specific gravities are met with in diabetes insipidus, waxy disease, chronic interstitial nephritis and cystic kidneys. A high specific gravity is met with when large quantities of urea, salts or sugar are present in the urine. It is often possible to say which of these conditions is present from the colour of the

urine. It is mostly pale when sugar, and high-coloured when urea or salts are present.

Urea.—The amount of urea in normal urine is about 2 per cent., *i.e.*, about 500 grains in the twenty-four hours, when an ordinary diet is taken. An excess of nitrogenous food or of liquid causes an increase, deficiency a decrease. Disease sometimes causes an increase, more frequently a decrease. The most marked increase is seen in diabetes mellitus. It is explainable in great part by the increased quantity of nitrogenous food and of liquid ingested, but when due allowance is made for them, some measure of increase generally remains, which is attributable to abnormal tissue waste. Hence the estimation of the urea output in cases of diabetes mellitus may serve as an important guide to the rapidity of their progress. There is an increase of urea also in fevers, not only a percentage increase (largely on account of the diminished quantity of urine voided), but an actual increase, allowing for the diminution in the ingesta, *i.e.*, a greater amount than is excreted by a healthy person on the same diet. Diminution in the quantity of urea excreted is seen in a large number of diseases; in all wasting diseases, particularly if associated with diminished appetite or vomiting, *e.g.*, cancer of the œsophagus or stomach; in many diseases of the liver, *e.g.*, cirrhosis, acute yellow atrophy (where it may be reduced even to nil); in all renal diseases in which there is loss of renal epithelium. In acute nephritis there may be, and usually is, an increased percentage of urea owing to the small quantity of urine excreted, but there is a real and generally great decrease in the total amount of the urea excreted in twenty-four hours. In chronic nephritis the amount of urea is mostly decreased, particularly if dropsy co-exist, but should the latter disappear, the amount of urea excreted may rise to, or nearly to, the normal. When a deficient amount of urea is excreted over long periods, as in renal disease, the amount present in the blood and tissues

increases. A sudden diminution in the quantity of urea excreted is frequently seen when uræmia is about to ensue.

URÆMIA.

This name is given to a group of symptoms indicating a toxæmia which arises in the course of many renal diseases. It is always grave, and frequently fatal.

Etiology.—Two theories are supported. The one holds that the toxæmia is caused by a diminished renal excretion, *i.e.*, that some substance or substances, usually excreted by the kidney, gradually accumulate in the blood and cause the symptoms chiefly by their action upon the nervous system. The toxicity of the blood serum in uræmia has been shown experimentally to be increased, but the nature of the poison has not been determined. The other view holds that the poison is the product not of diminished renal excretion but of an abnormal metabolism of unknown character. Some think that the kidney has an internal secretion whose disturbance may produce the poison.

Symptoms.—The onset may be sudden or gradual and the course acute or chronic. The symptoms are chiefly cerebral in the acute, and gastro-intestinal in the chronic varieties. The cerebral symptoms are coma, convulsions, mania, delusional insanity, paralysis, dyspnœa, sleeplessness, headache, &c. The commonest of all these is coma. The patient, with or without previous delirium or convulsions, becomes drowsy and gradually comatose. The coma may be preceded by headache, muscular twitchings, cramps, vomiting, or blindness. When coma has supervened, the pupils are usually contracted, the knee jerks excessive, the temperature subnormal, the tongue dry and brown, and the breath foul. This condition of stupor or coma may prove quickly fatal or last for weeks or even months. Mania is not common. Delirium, noisiness and restless-

ness are more frequent and usually give way to drowsiness and coma. Delusional insanity, particularly delusions of persecution, is sometimes met with. Convulsions, often identical with those of epilepsy or of Jacksonian epilepsy, occur. The fits may recur frequently and rapidly, resembling the "status epilepticus." The patient is unconscious, not only during the fits, but also in the intervals. Blindness (uræmic amaurosis) lasting for several days often follows the fits, or some of the other manifestations of uræmia. Paralyzes, such as hemiplegia or monoplegia, are not very uncommon, due, it is thought, to a localised œdema of the brain, since no gross lesion has been found after death. Dyspnœa is common. It may be continuous or paroxysmal. The attacks usually come on in the night and are often violent, causing the patient to sit up and gasp for breath. Sometimes the breathing is noisy and hissing, and frequently it assumes the Cheyne-Stokes type. Recovery may follow even after it has persisted for months, though usually it is followed by coma and other symptoms. Sleeplessness is sometimes very marked. It may be accompanied by hiccough and muscular twitchings. It often leads to a fatal issue, due to respiratory failure without the intervention of coma. Headache, often occipital, is frequent. Itching of the skin, frequently intense, and tingling of the fingers may be present. The gastro-intestinal are much more constant than the cerebral symptoms. They are nausea, vomiting and diarrhœa. They may come on suddenly and run an acute course. More commonly they are chronic. Vomiting is the most constant of them. It may persist for months. Occasionally diarrhœa comes on without vomiting. Nervous symptoms, particularly coma, usually supervene after a time, or some inflammatory complication, such as meningitis, occurs.

Treatment.—The treatment for uræmia will be given under chronic Bright's disease.

Uric acid, urates.—Uric acid is the chief member of the purin group of bodies found in the urine. The others are xanthine and hypoxanthine. The amount of uric acid excreted daily in the urine is about 10 grains. Its source, like that of other purins, is the nuclein derived from nuclear destruction in the food and in the tissues in about equal parts. In the urine it is combined with various bases (sodium, potassium and ammonium), of which sodium is the chief in the form of an acid sodium salt. If the urinary salts be deficient, as in dilute urines, the uric acid will not all be kept in combination, and that portion which is liberated will be deposited as a cayenne-pepper-like sediment when the urine cools. On the other hand, if the salts and pigment be abundant, as in concentrated febrile urines, the uric acid, even though it be present in a considerably higher percentage, will remain in combination, and there will be, when the urine cools, a brick-red-like deposit of urates which re-dissolves on heating. In either case the deposit rarely means an excessive formation and excretion. This does occur in splenic leukæmia, but it is doubtful if there is, as many assert, an excessive excretion after a gouty paroxysm, in fevers, in pernicious anæmia, malaria and some forms of diabetes mellitus (sometimes called gouty diabetes). Uric acid may form calculi. Lithæmia or the uric acid diathesis is a term introduced by Murchison to indicate a hypothetical condition of hepatic inadequacy induced by high living with deficient exercise, a sort of pre-gouty state which may pass on to true gout. The liver is probably the seat of conversion of nuclein into uric acid.

Kreatinin.—The amount excreted daily is about twice that of uric acid. It is chiefly derived from the food, but it is also derived from the tissues, since it is increased in fevers and in wasting diseases, where there is no marked diminution of metabolism. Its chief clinical importance lies in the fact that it, like urates when concentrated,

reduces copper, though only after boiling for a time, and may lead to a mistaken diagnosis of glycosuria. It agrees with uric acid, however, in not reducing alkaline bismuth solutions, and can thus be easily distinguished from sugar.

The urinary salts.—(1) *Oxalates.*—The oxalic acid excreted in the urine comes from the food. It is increased after the ingestion of foods rich in it, *e.g.*, rhubarb and other vegetables and many fruits. A persistent increased excretion and deposition of oxalic acid in the urine (oxaluria) is considered to be pathological. It is accompanied by symptoms of dyspepsia and nervous depression attributed to it by many, though regarded by others as due to some disturbance of metabolism of which the oxaluria is merely one of the signs. It is often present in the condition known as lithæmia. It may lead to the formation of calculi (calcium oxalate). (2) *Phosphates.*—Thirty to ninety grains (2 to 6 grammes) of phosphoric acid are excreted daily in the form of alkaline (potash and soda) and earthy (lime and magnesium) salts. The phosphates are derived largely from the food and partly from the tissues (nuclein, lecithin, &c.). Estimation of their amounts must be based upon the total excreted, and not upon the amount deposited. In febrile diseases the amount is diminished at first, but increased later. It is increased in conditions attended with destruction of leucocytes, in wasting diseases, acute yellow atrophy of the liver, and in leukæmia. Earthy phosphates may be deposited within the bladder and passed as a whitish fluid at the end of micturition. This may be mistaken for spermatorrhœa. It is mostly of no importance, but when associated with neurasthenia and a real increase in the excretion, it may indicate an abnormality in the calcium metabolism. The name of phosphatic diabetes is sometimes given to this condition, when there is also thirst, emaciation and polyuria. The phosphates may form calculi. (3) *Chlorides.*—Ten to fifteen grammes of sodium

chloride are excreted daily. The amount depends largely upon the amount in the food. The chlorides are diminished in fevers, particularly in pneumonia, where they may almost or completely disappear from the urine during the fever, to reappear again after the crisis. The chlorides are diminished also in diarrhoea. (4) *Sulphates* are present in small amount in normal urine. The most important are the ethereal sulphates which are composed of certain organic radicles, *e.g.*, phenol, indoxyl and skatol, combined with sulphuric acid. They are derived from putrefactive decomposition of proteids in the intestine, and hence are increased in intestinal obstruction, &c., though not in constipation. The urine containing them is colourless when passed, but after decomposition sets in, or when treated with an equal volume of hydrochloric acid and then adding a freshly prepared solution of hypochlorite of calcium drop by drop, so as to avoid excess, indigo (in the case of indoxyl sulphate of potassium or indican) is set free and may be extracted by adding a little chloroform, which dissolves it on shaking. This is spoken of as indicanuria. It causes the urine to become of a blue to a black colour. This is the commonest cause of black urine.

Pigments.—The pigment to which the colour of the urine is chiefly due is urochrome. Other pigments present are urobilin, uroerythrin and hæmatoporphyrin in small quantities. Both urobilin and hæmatoporphyrin may appear in considerable quantities in disease. Copious internal hæmorrhage and diseases associated with blood destruction, *e.g.*, pernicious anæmia, produce a large quantity of urobilin in the urine. It imparts the brown colour to the urine and the lemon-yellow colour to the skin and conjunctivæ which are so characteristic of pernicious anæmia. It is often mistaken for bile pigment, though it may be present along with bile pigment, as in cirrhosis of the liver. Hæmatoporphyrin may be present in such quantities as to impart a port-wine colour to the urine, as after the admin-

istration of sulphonal, in some cases of rheumatic fever, Addison's disease, peritonitis and hepatic cirrhosis. Uroerythrin imparts a pink colour to urinary sediments, e.g., uric acid. It is increased by high living, muscular activity, or fever.

Cystin.—This is a sulphur-containing substance, crystallising in flat hexagonal plates, which occasionally appears in the urine. Its importance lies in its tendency to form calculi, which may be very numerous.

Leucin and Tyrosin.—These substances, found normally in the alimentary canal, are invariably present in the urine in large quantities in cases of acute yellow atrophy. In phosphorus poisoning, on the other hand, though frequently present the quantities are small. They are also present sometimes in cases of hepatic cirrhosis.

ABNORMAL CONSTITUENTS.

These are proteids, carbohydrates and pigments.

The proteids are albumin, albumose and blood. When serum-albumin or serum-globulin, or both, as is almost always the case, are present, the condition is known as *albuminuria*; when albumoses are present as *albumosuria*; when blood is present as *hæmaturia*; and when blood pigment alone is present as *hæmoglobinuria*.

ALBUMINURIA.

This has until comparatively recently been generally regarded as a serious condition indicating disease of the kidneys. It is now known to occur, though comparatively rarely, at all ages in health, or at any rate without any discoverable change in the structure of the kidney. This occurrence is often called "physiological," though it might be more correctly called functional. It is most common in young adults, particularly after unusual muscular exer-

cise, *e.g.*, a football match, a meal rich in albumin, a cold bath or violent emotion. It may be absent in the morning and appear during the day, particularly after some muscular effort. When it appears at more or less definite intervals, it is called "cyclical albuminuria." In some cases it lasts only during adolescence, in others for many years. It is usually unaccompanied by tube casts. The quantity of albumin is usually small, but sometimes it is considerable. Albumin accompanied by hyaline and finely granular casts is not uncommon in apparently healthy adults, and the frequency increases with age. It is therefore generally regarded as an indication of senile change in the kidneys having little practical importance. The "neurotic albuminuria" seen after an epileptic attack, or in several nervous diseases, may be classed among the functional forms. Febrile albuminuria is probably on the border line between diseases without and diseases with gross renal lesions, for in most feverish conditions there is a slight and transient albuminuria, *e.g.*, in typhoid, pneumonia, measles, &c. It usually disappears with the fever, but in some, such as scarlet fever and diphtheria, there is a great tendency towards permanent renal changes. Toxic albuminuria in many of its instances may be placed in the same category. Thus lead, syphilis, anæmia, cantharides, turpentine, &c., may cause slight transient albuminuria, liable, however, to become permanent. In the latter case it becomes "pathological." Pathological albuminuria is that arising from renal disease, either mere congestion, as in cardiac disease, or pressure on the renal vessels by the pregnant uterus (congestive albuminuria), or organic disease of the kidneys, such as acute or chronic Bright, waxy degeneration, suppurative nephritis, &c. Purulent inflammations of the pelvis of the kidney, ureter, bladder or urethra produce albumin in the urine; but they also produce pus, and they are therefore usually included under cases of pyuria and not under cases of albuminuria. The albumin of the

urine is a varying mixture of serum-albumin and serum-globulin, the proportion of the former being usually much the greater. This is what one would expect owing to the size of the molecule of the former being smaller than that of the latter. In rare cases the amount of globulin may exceed that of albumin. It may even be present alone. A high proportion of globulin is usually held to mean severe disease of the kidney. Indeed, both on clinical and experimental grounds, there seems good reason to draw the following deduction from the relative proportions of albumin and globulin. The presence of albumin alone means slight damage of the glomerular epithelium only. When the albumin is accompanied by globulin the damage is greater and extends to the renal epithelium. The higher the proportion of globulin the greater the damage, so that when the damage is excessive the ratio of globulin to albumin may approximate to that of the blood. The presence of albuminuria has therefore a variable significance. It may mean (1) no departure from health, (2) only a slight and transient renal disease, (3) grave renal mischief; and inasmuch as the last is by far the most frequent, the physician should always be placed on his guard whenever albuminuria appears.

ALBUMOSURIA.

Proteoses, rarely peptones, occasionally appear in the urine, *e.g.*, in pneumonia and other febrile conditions and in chronic suppurations, *e.g.*, empyema. They are mostly formed by the pyogenic cocci or other infective agent and merely excreted by the kidney. They are not usually of any clinical importance, but they are met with in one remarkable and fatal condition associated with sarcomata or multiple myelomata of bones known as "myelopathic albumosuria" or Kohler's disease, first described by Bence Jones in 1848.

PYURIA.

Pus is present in the urine in a large number of inflammations affecting the urino-genital tract, *e.g.*, pyelitis, pyelonephritis, cystitis, urethritis, leucorrhœa, rupture of an abscess into the urinary passages, and in tuberculosis of the kidney or bladder.

CHYLURIA.

The urine is milky from the presence of chyle or fat. The former is met with in filariasis, and occasionally in other rare little-known conditions; the latter in pregnancy and diabetes.

Treatment.—The treatment, as a rule, consists in an attempt to relieve the causal condition present: it is not easy to suggest any very hopeful plan.

HÆMATURIA.

Blood may enter the urine from any part of the urinary tract, *viz.*, the kidney, ureter, bladder or urethra. When coming from the kidney it is usually intimately mixed with the urine, imparting to it a smoky, a bright or dark red tint according to quantity. But it may flow from the kidney into the pelvis or the ureter and clot there, being afterwards passed in the urine as irregular or cylindrical thrombi. When it comes from the urethra it appears at the beginning, when from the bladder usually at the end of micturition. The renal conditions producing hæmaturia are venous congestion, infarction, acute and chronic inflammations, tubercle, cancer, calculus, drugs—*e.g.*, carbolic acid—*filaria sanguinis hominis*, bilharzia and injuries. The urinary passages may be the source of the blood, as in cystitis, papilloma, tubercle or calculus of the bladder, bilharzia, urethritis and injury of the bladder or urethra. There are considerable variations in the amount and persistence of

the blood. In infarction it is abundant, comes on suddenly, lasts for two or three days and then disappears. In some cases of acute nephritis, calculus, malignant disease and papilloma of the bladder it is also abundant and persistent. In other diseases it is usually scanty.

Treatment.—The appearance of blood in the urine demands in the first place an attempt to discover the source of the hæmorrhage. Where the hæmaturia is renal in origin, and if it is due to acute inflammation of the kidney, rest is essential, and as far as possible the renal functions should be carried on by the skin and the bowels. The treatment, therefore, consists of diaphoretics such as the liquor ammonii acetatis, jaborandi and similar remedies, and also purgatives, such as magnesium sulphate. Should the hæmorrhage be the result either of a tumor in the bladder, or of a calculus in the kidney or bladder, the treatment is essentially surgical. The hæmorrhage associated with trauma, tubercle, bilharzia or filaria sanguinis hominis may be treated by the use of astringents or hæmostatics, but the amount of blood lost and the condition of the patient in each case must modify the plan of treatment adopted.

HÆMOGLOBINURIA.

This is distinguished from hæmaturia by the absence of red blood-cells and the presence of blood pigment only. The urine is smoky, red, brownish-red or blackish-red in colour. It occurs in all conditions in which there is solution of the red blood-cells, viz., in infective diseases, *e.g.*, septicæmia; after certain drugs, *e.g.*, quinine, chlorate of potassium (toxic); after severe burns or excessive cold or fatigue. The peculiar form attacking horses has already been referred to. A remarkable variety is the *paroxysmal* hæmoglobinuria, which is liable to recur after any exertion or chill, however slight. Dipping the hands or feet in cold

water may bring it on. It is often present in Raynaud's disease. Various theories have been advanced to explain the disease, *e.g.*, that it is nervous in origin, but none of them is more than speculative.

Treatment.—The treatment depends on the nature of each case; if it is *paroxysmal* then care should be taken to obviate chill or fatigue, and warm drinks of a non-alcoholic nature give considerable relief during the attack. In cases which are of *malarial* origin, quinine should certainly be administered, and in *syphilitic* cases iodide of potash. Where no satisfactory etiological factor can be discovered, rest in bed with warmth, simple dieting and possibly such a remedy as nitro-glycerine might be tried.

NUCLEO-ALBUMINURIA AND MUCIN.

A shiny deposit, generally spoken of as mucin, is met with in inflammatory conditions. Mörner regards it as being a combination of proteid with chondroitin sulphuric acid. This view allies it to waxy material.

THE CARBOHYDRATES.

Dextrose is the most important reducing substance met with in the urine. It has already been considered under "Diabetes Mellitus." Lactose may appear in the urine in pregnancy. Both these give the tests for sugar, but inosite, which may appear in diabetes insipidus, does not give them. It is important to remember that other substances than sugar may reduce cupric oxide, *e.g.*, uric acid, kreatinin, and glycuronic acid. The last named substance is present in the urine after the administration of certain drugs, *e.g.*, chloral and camphor, and in cases of indicanuria, for the indol and skatol formed in the alimentary canal appear in the urine partly as compounds of glycuronic acid. Acetone is found in the urine in diabetes mellitus, fevers, and malig-

nant disease. It is not the cause of diabetic coma, but it is the cause of the ethereal odour of the urine and breath noticed in many cases of diabetes. It is frequently accompanied by diacetic acid.

THE PIGMENTS.

Melanin or black pigments resembling it are occasionally passed in the urine. The urine may be coloured when passed, but it usually becomes brown or black shortly afterwards. The existence of melanuria is generally regarded as an indication of melanotic sarcoma, particularly of the liver. It is to be remembered that indicanuria is commoner than melanuria as a cause of black urine. *Alcaptonuria* is another cause of dark urine. Homogentisic acid is passed in the urine. Attention is generally drawn to it by the brown staining of the linen. It is a congenital defect in normal metabolism, of a family and hereditary character. It is harmless. In some cases the cartilages and ligaments are blackened and the skin of the face and hands, &c., may show blackish pigmentation. These cases have been described as *ochronosis*. *Bile*.—The bile pigments appear in the urine (choluria) in cases of jaundice and impart to it a colour varying from reddish-brown to almost black. On shaking the urine a greenish-yellow froth is formed. This can often be detected in cases of jaundice before the skin has become coloured. Urobilinuria may present a superficial resemblance to choluria, and hence Gmelin's test for the latter should always be employed.

TUBE CASTS.

The products of inflammation in the glomeruli or renal tubules usually form cylinders, which are generally spoken of as "tube casts." They may be so abundant as to form a definite deposit visible to the naked eye, but this is rare,

and their presence is usually detected by microscopically examining the deposit obtained after the urine stands in a deep conical vessel for a time, or better still after it has been centrifugalised. The casts vary in length and breadth, and the latter characteristic may enable the observer to say what part of the tubules they have come from. Their presence in the urine does not necessarily mean renal disease, but it often does so, and it is further often possible from a study of their characters to form a correct diagnosis of the nature and extent of that renal disease. Different names are given to them, according as they show a homogeneous, a granular, or a cellular structure. The homogeneous casts have been classified as hyaline, colloid and waxy, the hyaline variety being the most common. These hyaline casts are more or less transparent and are formed by the coagulation of proteid material exuded from the renal epithelium or the glomeruli. They are mostly composed probably of serum-albumin and globulin. It was formerly thought that they were composed of fibrin, but this is comparatively rarely the case. It only occurs in hæmaturia or in serious renal disease, since the molecules of fibrinogen are too large to allow of easy passage through the tubular or glomerular epithelium. Hyaline casts frequently accompany albumin in cases in which there is no gross lesion in the kidney, particularly later in life; but they are also present in some forms of renal disease, such as the granular kidney. Hyaline casts also form a basis for other casts. Being somewhat sticky they collect granules and cells of all kinds, and become converted into granular, fatty and epithelial casts. Colloid casts are less transparent, and usually of a pale yellow colour. They form laminated and concentric spheres and cylinders, and are chiefly seen in chronic interstitial nephritis. Waxy casts are the translucent, slightly yellow and brittle solid masses seen in the tubules in waxy degeneration of the kidney. They occasionally give the reactions of waxy material, and are thought

by many to be of a true waxy nature, though this is doubted by others. Granular casts are of two kinds, fine and coarse. The finely granular casts are formed by minute particles of a proteid nature, extruded from the renal cells, held together by a hyaline basis. They are often present when there is no obvious renal disease, particularly in old people, but they also occur in acute and chronic nephritis. The coarsely granular casts may be partly formed in the same way, but they are chiefly formed by fatty globules, which result from cellular degeneration. They have generally a hyaline basis. They mostly indicate renal disease, and are particularly characteristic of chronic parenchymatous nephritis. The fine granular casts are generally pale, and the coarse generally dark in colour. The cellular casts are formed either by the renal epithelium or the blood cells, with a binding basis of hyaline material. The renal epithelial casts may show the characteristic rodded cells of the convoluted tubules little or not at all altered; but more usually the cells show much granular and fatty change and disintegration. They occur chiefly in acute parenchymatous nephritis. The blood-cell casts are formed either of leucocytes or of red cells. The former indicates acute and often suppurative inflammation of the kidney, and the latter occur in both active and passive renal congestion, thrombosis, infarction, acute nephritis, and in some forms of chronic parenchymatous nephritis.

CALCULI—NEPHROLITHIASIS.

Etiology.—Calculi are more common in men than in women. They occur at any age, but are especially frequent in youth. They are due either to disturbances in the metabolic processes of the body or to conditions influencing the crystallisation and deposition of the ingredients of the calculi. A little blood, mucus, pus or albumin may form a nucleus for the formation of a calculus.

Composition.—They consist of either uric acid, oxalate of lime, phosphate of lime (sometimes mixed with carbonate), of a mixture of uric acid and oxalates (often in alternate layers), or of cystin. Uric acid calculi are the commonest. As already stated, uric acid has a double source, viz., the food and the tissues. The disturbance in metabolism which leads to an excess of uric acid may occur, then, either in the food or in the tissues. The former can be largely or completely controlled by giving a diet free from purin or alloxuric bodies. Further, the amount of urinary salts greatly influence the crystallisation of uric acid, hence dilute urines poor in salts tend to deposit free uric acid, even when there is no disturbance of metabolism leading to an excess of formation. This lack of salts is asserted to be the cause of the frequency of calculi in India and China. The uric acid calculus consists either of pure uric acid or of urates. The pure uric acid calculus is usually small (a pea to a pigeon's egg), of a colour varying from yellow to brown, of a rough surface, and of a brittle consistence. The urate calculus consists of urates of ammonium and magnesium and usually has an admixture of uric acid and phosphates. They are generally larger (about the size of a hen's egg), smooth and pale, though their characters vary according to the predominance of particular urates. The oxalate calculi have a very dense consistence, an irregular shape, and a brownish colour. They are associated with gastric disorders, particularly indigestion and dilatation of the stomach. The acid phosphate of soda normally present in the urine prevents the formation of oxalate of lime, and hence oxalate calculi, like uric acid calculi, tend to occur in dilute urines. Phosphatic calculi consist of phosphate of calcium, ammonium magnesium phosphate or carbonate of calcium. They vary in size, being often large. They are of a white or grey colour, and are either hard, or more frequently soft and crumbling. Cystin calculi are uncommon. They are soft and

brownish, and generally small in size. They may be very numerous.

Position of the Calculi.—Calculi are most frequently present in the pelvis and calyces of the kidney. Occasionally they are found in the kidney substance, usually the medulla.

Results of nephrolithiasis.—When the precipitated salts occur in a molecular form, they may lodge in the tubules or pass out in the urine as a visible granular sediment, a condition which is spoken of as *gravel*. It is of slight significance in itself. When calculi are formed in the kidney substance they may become encapsulated and give rise to no trouble. In other cases, and generally when present in the pelvis or calyces, they predispose to pyelitis, pyelonephritis and abscess of the kidney.

Treatment.—For an attack of renal colic the treatment consists in the application of fomentations over the abdomen, with the administration of morphia, and sometimes, where the pain is excessively severe, chloroform anæsthesia. It is usually advisable to make the patient drink large quantities of water, in certain cases alkaline mineral waters, so as to help the stone in its passage downwards. The further treatment of stone in the kidney, and certainly the treatment of stone in the bladder, belong to the domain of surgery; but it must not be forgotten that prior to an operation on one kidney the functioning capabilities of the other kidney should be ascertained, and the use of Luy's separator enables the physician to decide as to the adequacy of each kidney by itself. There are many baths abroad where patients suffering from uric acid or oxalic acid calculi have passed them during or after a course of the waters, and it is certainly desirable in cases in which uric acid and oxalates are present in the urine to endeavour to rectify the chemical error present. It is in this way that alkaline mineral waters have been found of most

efficacy. It is, however, practically certain that no solvent of uric acid can be administered to a patient in a dose capable of acting efficiently during its excretion in the urine. A remedy often given as a solvent of uric acid is piperazine in doses of 10 to 20 grains thrice daily.

INFLAMMATION OF THE KIDNEY— NEPHRITIS.

There is a considerable difference in the meaning attached to the terms *nephritis* and *Bright's disease* by various writers. Some regard them as synonymous, as applied to both acute and chronic inflammations of the kidney. Others look upon nephritis as the wider term, including all forms of inflammation of the kidney, whether slight or severe, and use Bright's disease only for the chronic malady, or at most extending it to the severe forms of the acute disease characterised by marked renal changes and prominent clinical symptoms, notably albuminuria and dropsy. Nephritis is the better term from a purely pathological standpoint, but not from a clinical one. The minor forms of nephritis give rise to no definite clinical symptoms during life, and hence it is convenient to use the term Bright's disease for the major forms of nephritis giving rise to clinical symptoms capable of detection during life.

The general etiology of nephritis is easy enough. It is always caused by a poison. The origin of this poison may be threefold—(1) it may be ingested, (2) it may be the product of abnormal metabolism, or (3) the product of an infective process. Alcohol and lead are the best known among the poisons of the first group. They are probably capable of causing only the chronic form of the disease. Gout stands foremost in the second group; while the infective fevers and infective processes generally, such as

scarlet fever, diphtheria, measles, syphilis, &c., constitute the third (by far the largest) group. On the other hand, the classification of the varieties of nephritis, either on a pathological or a clinical basis, is full of difficulty. Many refinements have been attempted, but it seems unnecessary to give an account of them here, for the practical physician is chiefly concerned with the correlation between morbid changes and clinical phenomena, and seeks a classification which brings them into line as far as possible. The tubules, the glomeruli and vessels generally and the interstitial tissue constitute the kidney structures, and any inflammation of the organ, whether it be acute or chronic, affects all three, though it may involve them unequally in different instances. This difference in degree of involvement may be so great that a differentiation into tubular or parenchymatous, glomerular, and interstitial nephritis becomes more or less possible histologically, though rarely clinically to the same degree. The nephritis due to cantharides is mainly glomerular, and the same is true of many cases of nephritis due to scarlet fever. The heavy metals produce an inflammation, degeneration and necrosis of the epithelium of the convoluted tubules, while many animal and vegetable poisons, *e.g.*, snake venom, eel's blood, ricin, &c., produce a similar, though less severe, action, since the degeneration does not go on to cell necrosis. The same change is seen after the administration of vinylamine, an amine of vinylic alcohol, but it is combined with considerable glomerular change. The nephritis is acute when the doses are large, chronic when they are smaller and more prolonged. The poisons of microbic origin probably mostly act much in the same way as vinylamine.

ACUTE NEPHRITIS—ACUTE BRIGHT'S DISEASE.

Etiology.—I. THE INGESTED POISONS.—Turpentine, cantharides, carbolic acid, the metallic poisons, and a large

number of organic poisons, such as snake venom, ricin, eel-serum, &c., may set up an acute nephritis.

2. THE METABOLIC POISONS.—They are little known, and hence it is difficult to estimate their influence in the causation of acute nephritis.

3. THE INFECTIVE PROCESSES.—*Cold*.—A chill has preceded many attacks and has been blamed for them and rightly, for though cold is not in itself capable of setting up the nephritis, it so lowers vitality that a circulating poison brought to the kidney is able to do so. *Pregnancy* stands in some similar relationship. Pressure upon the renal veins is thought by some to be the way in which it causes the nephritis, but it is much more likely that in pregnancy, as in chill, poisons at present undetermined may be produced to which the nephritis owes its origin.

Infective fevers.—They present no difficulty. The poisons which cause the specific fevers are the causes of the nephritis. They circulate through all the tissues of the body, but their influence upon the kidney may be greater than upon the other organs of the body, owing to greater concentration during their excretion by the kidney. Hence it is particularly in these infective fevers, where the elimination of the poison is in whole or in large part by the kidneys, that acute nephritis is common. Thus it is frequent in scarlet fever, in diphtheria, and in cholera: less frequent in typhoid fever, measles, small-pox, malaria, pneumonia, meningitis, &c. It may arise in a great many other infections, such as syphilis, tubercle, rheumatism, pyæmia and septicæmia, and acute tonsillitis. The kidney excretes not only the microbic poisons, but also the microbes themselves, and it is to the former rather than to the latter that the nephritis is due. Thus the typhoid and the tubercle bacillus and others may appear in the urine without the kidneys showing any grave structural change. Their filtering power is impaired and the bacilli are allowed to pass, but there is no evidence of any nephritis. When, however,

the germs reach the kidney in sufficient numbers to block any of the capillaries, they usually produce their characteristic lesions. Thus in tuberculosis of the lungs there may be renal tubercles, and in malignant endocarditis or any other pyæmic disease there may be renal abscesses.

Morbid Anatomy.—The kidneys are mostly swollen, the increase in thickness being particularly noticeable. The capsule is unaltered and strips off readily. The kidney surface is pale, of a greyish-red colour, and mottled with red and a few pale yellow points. Sometimes, particularly in early stages, it is deeply congested, but in all cases it shows the red or dark red points characteristic of distended vessels or perivascular hæmorrhages. On section the same changes are seen in the cortex, particularly in its superficial part. It is to the swelling in this situation that the increased size of the organ is mainly due. The red points may be hæmorrhages or distended glomeruli. The latter may stand out prominently owing to their congestion and increase in size, or they may be pale and difficult to see. The medullary portions of the pyramids are deeply congested and their dark red colour is in marked contrast to the pale greyish-red of the cortex. This pallor is more marked in the later stages, and it becomes mottled with yellow from fatty change in more advanced and prolonged cases. This description applies to the forms of acute nephritis usually met with as the result of some infective process, but in very mild cases, where the nephritis is a transient affection occurring only during the height of the fever and passing away during convalescence, the change may be so slight as to produce no change visible to the naked eye; and even in more severe cases, where the glomeruli or the interstitial tissue and not the tubules are mainly affected, there may be little to be seen beyond a little enlargement of the glomeruli and of the cortex generally. Microscopically, the changes, though varying greatly, are naturally arranged

in three groups—(1) *Tubular changes*.—The cells lining the convoluted tubules lose their striation, become swollen and granular, and hyaline and fatty droplets appear in them. They may proliferate, but in most cases they are shed in part, sometimes *en masse*, into the lumen, which is also charged with exudate, leucocytes and red blood-cells from the intertubular capillaries. The tubules thus become dilated in places, owing to these products obstructing the flow of urine. This is the chief cause of the swelling of the organ as a whole. Similar, though much less marked, changes are seen also in the other tubules, except that epithelial, blood and hyaline casts are more common in the collecting tubules, owing to their being washed down from above. (2) *Glomerular changes*.—The lumen of the capillaries may contain hyaline thrombi and numerous cells, which are also to be seen packed more or less densely around the capillaries. These cells come in part from proliferation of the endothelial cells of the capillary walls, and in part from emigrated leucocytes. The epithelium lining the glomerular tuft on the outside and Bowman's capsule on the inside may proliferate and fill the capsular space in whole or in part with cells. These cells are washed down into the tubules, and the capsular space may also contain blood or coagulated albumin. The periglomerular tissues are often crowded for some distance with similar cells to those of the glomerular tufts themselves. (3) *Interstitial changes*.—These are in many cases restricted to a serous exudate containing red and white cells, but in some cases there is also a small-celled infiltration diffusely spread, or restricted to certain areas, particularly to the periglomerular regions as above mentioned. When these interstitial changes are the chief morbid changes present, the disease has been called "acute interstitial nephritis." It has been described as occurring particularly in children after diphtheria, scarlet fever and measles.

Symptoms.—In the majority of cases of the milder forms of acute nephritis there are no symptoms, with the exception sometimes of pains in the back. In the more severe forms, usually spoken of as acute Bright's disease, there are well marked, though variable, symptoms. The onset is usually slow, the patient complaining of pains in the limbs, nausea and lassitude, with puffiness of the face and ankles. It is sometimes acute, particularly after cold, when general dropsy may become severe, even within twenty-four hours. Fever is unusual, except in children, in whom a temperature of 101° to 105°F. may exist for a few days. Convulsions may be the first symptom of the mischief in pregnant women and in children, particularly in scarlet fever cases. Attention is drawn sooner or later to the changes in the urine, the most characteristic of all symptoms. The quantity is diminished, usually amounting to from 10 to 20 ounces, though complete suppression may exist, particularly at first. It is high-coloured, smoky to dark red, and deposits an abundant sediment on standing, which contains epithelial, blood and hyaline tube casts and red blood-cells. Albumin is usually abundant, though it may be slight or even absent for a time in rare cases. The total excretion of urea is diminished, but its percentage composition is usually increased, owing to the diminished quantity of urine excreted. Apart from the changes in the urine, dropsy is the most striking of the symptoms. It is present as a general anasarca, particularly noticeable in the face, ankles and legs. While subject to considerable variations, it is rarely so great as in chronic Bright's disease. It may extend to the pleural cavities even while it is slight in the legs. It rarely invades the lungs or the glottis. (In chronic Bright's disease oedema glottidis is comparatively common). It may be completely absent even when the nephritis is intense and the urine loaded with albumin and blood. The dropsy of acute Bright's disease is generally regarded as being toxic in origin. The circula-

ting poisons are supposed to injure the epithelium of the vessels, causing them to become more permeable. The amount of the dropsy stands in close relationship to the amount of urine excreted. As the former diminishes, the latter increases, until it reaches a higher amount than normal, when recovery is about to set in. The other symptoms of acute Bright's disease are also considered to be of toxic origin. Anæmia develops early and rapidly in most cases. Gastro-intestinal symptoms are nearly always present. They are nausea, hiccup and vomiting. Uræmic symptoms of varying severity occur in a certain number of cases. Dilatation of the heart may come on rapidly and cause sudden death. Papillitis, hæmorrhagic and albuminuric retinitis are much rarer than in chronic Bright's disease. Inflammatory complications, particularly of the serous membranes—*e.g.*, peritonitis, pericarditis, &c.—are very likely to occur during the course of acute Bright's disease.

Diagnosis.—The characters of the urine, the dropsy, and other symptoms make the diagnosis easy in most cases. The presence of albumin and casts alone do not, as already stated, always signify an acute nephritis, but when accompanied by dropsy, &c., such a diagnosis may be safely made.

Prognosis.—In the great majority of cases, particularly those following a chill, the symptoms gradually subside and disappear within a month or more. Occasionally there are several recurrences before recovery is complete. In some instances, though the symptoms subside they do not disappear, and the disease becomes chronic. Many cases prove fatal, particularly among the scarlatinal forms and among young children, in whom the mortality generally ranges from about ten to thirty per cent. of the cases.

Treatment.—The patient should be placed in bed and kept there until both albumin and blood have disappeared from the urine. He should be ordered milk diet, and the functions of the kidneys should be relegated so far as possible to the skin and bowels. There is, however, no objection to permitting the patient to drink freely of distilled water, which is certainly the simplest and least irritating of all diuretics. He should be clad in a flannel nightdress, and in cold weather should be placed between blankets. The skin can be made to act either by applying hot bottles round the patient's body or by giving a hot-air bath, and certainly in the case of children by an ordinary hot-water bath. If this treatment is begun early and the patient is willing to submit to the limited dietary, the results in an acute case are often extremely favourable. Unfortunately the milder forms of the disease may give rise to no suggestive symptoms which make the patient seek advice and so timely interference is prevented.

Pain in the back may be relieved by linseed poultices and sometimes by cupping over the kidneys. If uræmic phenomena develop, pilocarpin should be administered hypodermically in doses of $\frac{1}{12}$ th to $\frac{1}{8}$ th of a grain, and it must be remembered that the nausea, vomiting, severe headache, and in fact most of the manifestations of acute nephritis are of uræmic origin. In every case order purgatives freely, and in many instances every day. The simplest remedy is certainly magnesium sulphate in one to four drachm doses, but in many cases compound jalap powder in 20 grain doses will be found a useful alternative. The heart and pulse must be carefully and periodically examined, and where there is any cardiac dilatation, either strophanthus or digitalis should be administered. When the blood and also the albumin have completely disappeared from the urine the diet may be cautiously increased by the addition of arrowroot and milk puddings and eventually fish, but only very gradually should

red meat be resumed. The sequelæ of Bright's disease may call for special treatment, and in particular anæmia should be relieved by giving iron freely. Tincture of the perchloride of iron is a favourite remedy in many instances, both from its effect in curing the anæmia and also from its astringent properties. The after-treatment of a case of Bright's disease depends to some extent on whether the causal condition was an infective fever or whether it was the result of exposure to cold and wet. In every case, however, the patient should be guarded from the risks of another attack, and it is certainly of great advantage, where it is possible, to send a Bright's case away to a warm equable climate during the colder months of the year.

CHRONIC NEPHRITIS—CHRONIC BRIGHT'S DISEASE.

Chronic nephritis occurs in many different forms, but in all of them there are changes in the tubules, glomeruli and interstitial tissue; and they may consequently be divided with fair accuracy into a chronic parenchymatous and a chronic interstitial form.

CHRONIC PARENCHYMATOUS NEPHRITIS.

Etiology.—It often follows upon acute nephritis, but may be chronic from the first. In the latter case cold, syphilis (the secondary stage particularly), alcohol, malaria, tubercle, pyæmia, or the infective fevers may be the chief agent. It is met with mainly in young adults.

Morbid Anatomy.—The kidneys are usually increased in size to a moderate extent, rarely to a great extent (twice the normal or more). The capsule is thin and strips readily, leaving a smooth, pale surface in which the stellate veins stand out very prominently. On section, the cortex is swollen, opaque and yellowish-white in colour, and the

glomeruli may be enlarged. The pyramids are usually deeply congested. Microscopically, the epithelium of the convoluted tubules, and to a less degree of the looped and collecting tubules, show extensive granular and fatty degeneration, desquamation and disintegration. The tubules are dilated and contain many epithelial and fatty casts, occasionally even large quantities of blood (chronic hæmorrhagic nephritis). These changes are unequally distributed and do not affect all the tubules, some being almost normal. The glomeruli present considerable variations. They usually show hyaline change in the capillaries, cellular increase in the tuft and in the capsular epithelium. Occasionally the glomerular are far more marked than the tubular changes, particularly in cases arising during convalescence from scarlet fever, and these cases have been described as subacute or chronic glomerular nephritis. The interstitial tissue is everywhere increased, though not to a conspicuous extent. This kidney is often called *the large white kidney*. As time goes on a gradual diminution in size of the kidneys takes place, until they become even smaller than normal. This is due to a progressive increase in the interstitial tissue and a consequent shrinkage in the other structures. This constitutes what is known as the *small white kidney* and also as the *pale granular kidney* owing to its surface being rough and granular-looking. There is no doubt that the small white kidney often follows upon the large in this way, but it often arises independently as a primary form. In a well-marked case, besides the diminished size of the organ, the thickened and adherent capsule, the pale mottled and granular surface, there is much diminution of the cortex, which presents many yellowish spots and is always sharply distinguished from the medulla. Microscopically, the chief feature is the great increase in the interstitial tissue, which in places may show only a few atrophied glomeruli and shrunken tubules. Elsewhere throughout the kidney many of

the glomeruli show fibroid capillaries and greatly thickened capsules, and many of the tubules are collapsed and bereft of their epithelium, or possess an epithelium in an advanced stage of fatty degeneration, or are dilated into cysts. The blood-vessels of the body generally may be thickened and the left heart hypertrophied.

Symptoms.—When it arises as a sequel of acute nephritis the symptoms of the acute gradually merge into those of the chronic disease. In cases arising independently, there is a history of vague ill-health, or the patient may complain of dyspepsia or pallor, with puffiness of the eyelids or feet. In other cases, uræmic symptoms appear suddenly, or epileptic attacks occur. The condition of the urine is most important. It varies in the *large white* and in the *small white* kidneys. In the former it is scanty, highly albuminous, rich in tube casts (epithelial, fatty, granular and hyaline), in leucocytes, and sometimes also in blood. In the latter it is more abundant, highly albuminous, less rich in tube casts and in leucocytes, and rarely contains blood. The amount of urea is diminished in both. There is also considerable difference in the other symptoms. In the large form there is generally considerable and persistent dropsy, not only of the subcutaneous tissues, but also of the serous cavities, there is marked pallor and anæmia, usually nausea, vomiting, and not infrequently intractable diarrhœa, and the pulse shows signs only of slight or moderately increased tension. In the small form there is little or no dropsy, marked anæmia and pallor (the complexion being spoken of as *earthy*), much wasting, and sometimes marked pigmentation of the skin, so great at times as to suggest Addison's disease. The pulse shows the characteristic signs of increased tension, and the heart of left-sided hypertrophy. Uræmia is liable to occur in both forms, but more so in the small than in the large form; and the

same is true of ocular symptoms and of the liability to inflammatory complications, such as pericarditis, pleurisy, pneumonia, and they, particularly the first, are prone to run a latent course.

Prognosis.—Recovery is rare. It occasionally occurs in children after the disease has lasted for one to two years. While complete recovery is rare, partial recovery or great improvement under appropriate treatment is common, and patients may live for five, ten or even twenty years in comparative comfort. Death may, however, occur at any time, and sometimes with great suddenness, from cerebral hæmorrhage, uræmia, cardiac failure, or some complication.

Treatment.—The treatment closely resembles that already described for acute nephritis. The patient's diet should as far as possible be limited to milk; the anæmia from which every patient affected with this disease inevitably suffers should be treated by the free use of iron, and where there is any suspicion that a syphilitic element is present potassium iodide must be given. Dropsy is treated on the lines described under acute nephritis. Diuretics, such as potassium acetate, have often to be administered freely.

CHRONIC INTERSTITIAL NEPHRITIS.

Syn. Granular Kidney, Contracted Kidney, Cirrhosis or Sclerosis of the Kidney.

INTRODUCTORY.—These different terms may be regarded by the clinician as synonymous, although the pathologist may assign differences to them. They express a prominent pathological change presented by the kidney: thus granular applies to its rough surface, contracted to its smaller size, and in most cases the kidney is both granular and distinctly smaller than normal, though in some it is granular without

being smaller. Cirrhosis or sclerosis of the kidney or chronic interstitial nephritis brings into prominence the marked increase in the interstitial tissue which is seen in all varieties, both larger and smaller, of the disease. It is a chronic renal disease which, notwithstanding the differences in its mode of production, invariably runs a definite and similar clinical course, and finally results in the same morbid changes within the kidney.

Etiology.—(1) It may arise as a sequel of the large or of the small white kidney. They practically run into one another, both clinically and pathologically, in the later stages. (2) It may arise as an independent primary affection. (3) It may follow upon arterial sclerosis. This last variety should be restricted to cases which run a course clinically distinctive of chronic renal disease, in which the sclerosis of the renal vessels is primary and of a greater degree of development than that of the vessels of the body generally, and not extended, as is done by some authorities, to include cases of comparatively slight renal sclerosis due to the renal vessels participating, often only to a moderate degree, in a general widely-spread and highly-advanced general arterio-sclerosis. In the first section the causes at work have already been mentioned, but those of the second and third sections require at least a short consideration. It is scarcely possible, however, to specify separately those which act under each. They can be grouped under the same three heads as before, viz.—(1) poisons taken in by the mouth, (2) poisons which are the products of abnormal metabolism, and (3) poisons which result from bacterial action. Alcohol and lead are the most prominent examples under the first head, under which also may be placed rich foods, particularly an excessive use of meat. This last may come partly under the second head, as it leads to functional disturbance of the liver and an excess of uric acid and other purin

bodies, *vide* lithæmia. Gout is the best known cause operating under the second head, and the granular kidney is so often found in association with gout that it is frequently known as the *gouty kidney*. Syphilis in its tertiary stage is perhaps the best known of the bacterial causes. Several factors may operate together in some cases. In the independent primary form of the disease they act upon the tubular and other delicate epithelial structures (*e.g.*, the glomeruli) of the organ, causing their degeneration and atrophy, in consequence of which the intervening interstitial tissues undergo compensatory hypertrophy. In the arterio-sclerotic form, the smaller branches of the renal artery undergo thickening of their inner and middle coats, with progressive diminution of their lumen. There is thus a progressive loss of nourishment in the areas of renal tissue supplied by these vessels, which results, as before, in degeneration and atrophy of the more delicate and more highly specialised renal epithelium, with compensatory hypertrophy of the interstitial tissue. In some cases the cause operates both directly upon the epithelial tissues and indirectly upon them through the blood-vessels. In both, the process is a degenerative and not an inflammatory one, whereas in the first section, *viz.*, where the disease arises as a sequel to acute or chronic parenchymatous nephritis, it is essentially inflammatory in nature. All three ultimately result in the production of a kidney which shows the same morbid changes, both macroscopically and microscopically, so that it is generally impossible, from the histological characters alone, to say by which of the three methods it has been originated and evolved. Before leaving the question of etiology, a word should be said as to age and sex. It is about twice as common in men as in women, and while it may prove fatal at any age from the fifth year or under to the seventeenth year or over, it is rare under twenty, most common in middle life (forty to fifty), and

prevalent in later years. Senility and premature senility are strong predisposing factors. Heredity seems also to have some influence, particularly in cases which have occurred in children and young adults. There are a few recorded instances where several children of one generation have been its victims.

Morbid Anatomy.—Both kidneys are affected in a nearly equal manner. They are usually smaller than normal, sometimes reduced more than half their natural size, though in cases of inflammatory origin they may be even a little larger than normal. The capsule is thickened and adherent. The surface of the kidney is rough or granular, being studded with little hemispherical projections, and usually also with minute cysts. The colour is reddish, sometimes dark red, particularly in the gouty kidney. It is deepest in the sulci around the projections. It is often pale and mottled with yellow in cases of inflammatory origin. Section of the organ is made with some difficulty, as the substance is hard, firm and resistant. On section, the superficial cortex is seen to be diminished in thickness. The diminution is greatest under the sulci, where the whole thickness of the superficial cortex may not be more than one-sixteenth of an inch. It may show minute cysts in varying number. The deep cortex is similarly affected, though to a less degree. The medullary portions of the pyramids are atrophied, though not nearly so much as the cortex. There is a greatly increased amount of fat around the calyces, compensatory to the diminution of the renal tissue. The vessels, particularly the smaller branches of the renal artery, are thickened and stand out prominently. Microscopically, the cortex shows an alternation of dense and opener areas of renal tissue. The former lie around the interlobular arteries, underneath and continuous with the sulci on the surface, and the latter underneath the intervening projections. The dense areas show a fibrous

tissue, which in places gives no trace of either glomeruli or tubules, in other places clusters of altered glomeruli but no tubules are to be seen, and in others, particularly towards the margins of the dense areas, both altered tubules and glomeruli appear. The denser thus pass gradually into the opener areas. The glomeruli, which persist much longer than the tubules, show alterations, varying much in degree, in their tufts and in their capsules. The capsules may show much fibrous or hyaline thickening which compresses the tuft, or it may be compressed by a cystic dilatation of the capsular space, or the tuft itself may be converted more or less completely into a fibrous and hyaline knot. Clusters of such glomeruli may come to lie close together owing to the disappearance of the intervening and more delicate tubules. The glomeruli towards the central parts of the denser areas show the greatest degree of change, but even in the opener areas they are rarely quite healthy, usually showing at least some hyaline degeneration or cellular proliferation in their capillary walls. There may be no trace of tubules in the central parts of the denser areas, but towards their margins they can usually be detected in places as small spaces lined by a flattened or a cubical epithelium. They get larger in the opener areas, where they are frequently dilated into cysts. The epithelium lining them shows hyaline, granular or fatty degeneration, and their lumen contains hyaline, coarsely-granular or fatty tube casts. The intertubular capillaries lying between the tubules become impervious with the disappearance of the tubules. This interferes with the free emptying of the glomeruli, and leads to increased blood pressure therein. This in turn increases the watery discharge from the glomeruli, which may account for the polyuria usually present in this disease. The fibrous tissue increase is chiefly confined to the dense areas of the cortex, but it may be more or less diffused to a small extent in the pyramids. The renal arteries show marked

thickening of their inner coats, though the middle and outer coats are usually also sclerosed.

Associated changes in other organs.—Changes are generally to be found in nearly all the organs of the body, but some of these are more important than the others, notably general arterio-sclerosis, cardiac hypertrophy, hæmorrhages and inflammations. The relation of general arterio-sclerosis to chronic renal disease is one upon which there is much doubt. The particular form of it which appears after the renal disease in point of time may be regarded as caused by the renal disease, through the agency either of some circulating poison or of increased vascular tension, or of both. The hypertrophy of the heart is generally attributed to the increased work entailed by the heightened vascular tension, not only in the general arterial system but also in the kidneys themselves. It is practically constant, though it varies in degree, and while it usually affects the left ventricle chiefly, it may involve the whole heart. Hæmorrhages are very prone to occur. The brain is their most frequent site, a considerable proportion of apoplexies having this explanation. Its substance suffers much more frequently than its meninges. The retina is another common site, the hæmorrhages being most frequently flame-shaped, other situations being the nose (epistaxis being sometimes a very early symptom), the pelvis of the kidney, the uterus, and the stomach. Among the inflammations, bronchitis comes first in frequency, and next to it is pericarditis (the latter occurring as a complication of granular kidney more often than it does in any other disease except acute rheumatism), whilst pneumonia and pleurisy are about equally frequent.

Symptoms.—Latency is a leading feature of interstitial renal disease running a chronic course from the beginning. In a great many cases there are no symptoms until some serious complication, such as pericarditis, apoplexy or uræmia, suddenly appears. In others, indefinite symptoms

arise which may be attributable to the heart, such as shortness of breath, palpitation, &c.; to the general nutrition, such as lassitude and loss of strength, diminished appetite, furred tongue, wasting, &c.; or to the nervous system or eyes, such as headache, sleeplessness, failing or defective vision. The symptoms which are more or less characteristic of the disease may be grouped under the following divisions—urinary, circulatory, nutritional, and nervous.

The urinary symptoms.—The amount of urine passed in the twenty-four hours is usually increased, sometimes even double the normal but oftener about a half more than normal. Micturition is thus more frequent, and the patient may complain of having to rise once or oftener during the night. The colour of the urine is pale, and its specific gravity low (a persistent low specific gravity is a common feature). Albumin is present in small amount, though it may be absent at times. It is never large in amount, as it so consistently is in the chronic parenchymatous nephritis. Albumosuria is frequent. Blood is not uncommon, and occasionally the quantity may be large. Casts of the granular and hyaline variety are generally present. The amount of urea, as of other solid constituents, is generally, though not always, diminished.

The circulatory symptoms.—The pulse is hard and incompressible. This persistent high tension is an early and important symptom. The vessel wall is usually also thickened. The left ventricle (and later the whole heart) is hypertrophied. The second sound in the aortic area is accentuated, and the first sound at the apex may be reduplicated, or there may be a systolic murmur. After a time, cardiac dilatation and insufficiency may develop, producing a gallop rhythm, dropsy, and other signs of cardiac failure. Dropsy is not common and rarely extends beyond a little œdema about the ankles. It is practically always of cardiac and not of renal origin. At times it is serious, when sudden œdema of the glottis or of the lungs occurs.

Hæmorrhage from the nose is not uncommon. Shortness of breath, dyspnœa, pain and palpitation are other cardiac symptoms.

The nutritional symptoms.—Loss of appetite, of strength and of flesh are common. Vomiting and diarrhœa may be severe. Anæmia becomes marked, the skin assuming an earthy and somewhat cachectic appearance.

The nervous symptoms.—Headache and neuralgias are frequent. Cerebral hæmorrhage is especially apt to occur and frequently proves quickly fatal. Uræmic symptoms of varying degrees of severity are frequent. The ocular symptoms are particularly important. Diffuse retinitis or papillitis and retinal hæmorrhages are common. Amaurosis, independent of retinal changes, may occur. It is spoken of as “uræmic amaurosis.”

Diagnosis.—The characters of the urine, pulse and heart are the best guides, but they are not sufficiently marked in the early stages of the disease to make a diagnosis possible. When prominent cardiac symptoms are present, it may be difficult to distinguish cases of granular kidney from those of primary mitral disease; but careful attention to the history, the urine, the pulse, &c., over a period of time, if necessary, will generally enable it to be done.

Prognosis.—The measure of health and the duration of life are both most uncertain. Serious uræmic, cardiac, hæmorrhagic or inflammatory changes may occur at any time and suddenly and quickly prove fatal. Marked albuminuric retinitis is very unfavourable, the patient rarely living many months after its appearance. Numerous patients live for many years, enjoying fair health and comfort.

Treatment.—In deciding upon the treatment suitable for any particular case, the capacity of the kidneys should

be gauged and the evidences of disease carefully noted. These evidences include the presence and amount of albumin and the variety of tube-casts in the urine. In many instances it is only requisite to regulate the life and work of the patient, guarding him from mental worry and physical overstrain. An equable climate is desirable, and a word of caution should be given to the patient with regard to the use of alcoholic stimulants. It is not necessary to restrict the patient to a rigid milk diet, but the amount of butcher meat taken each day should be limited.

The treatment, otherwise, consists largely in combating the special conditions with which chronic nephritis may be associated. *High intravascular pressure* should be reduced by the free use of saline purgatives, by the judicious administration of drugs belonging to the nitrite group, and by the bath treatment which not merely lowers blood pressure but, in addition, stimulates diaphoresis. The most suitable baths are ordinary warm water baths, Turkish and radiant-heat baths, but the two latter demand careful supervision. *Anæmia*, so often present in patients suffering from all forms of Bright's disease, requires the administration of iron.

The treatment of *uræmia* in its acute form has been already referred to in connection with acute nephritis, and its appearance may call for prompt attention in granular contracted kidney. For chronic uræmia free purgation is desirable, and saline purgatives, such as magnesium sulphate, are of great value. Nitrites may also be administered, and especially in the form of nitro-glycerine ($\frac{1}{100}$ th grain) or nitrite of soda (2 to 5 grains). Turkish and radiant-heat baths, both of which induce very free diaphoresis, are beneficial and may be combined with the internal administration of jaborandi or a hypodermic injection of pilocarpin ($\frac{1}{12}$ th to $\frac{1}{8}$ th grain). There is no remedy which controls the delirium of uræmia like morphia.

The surgical procedure of stripping the capsule of the kidney has been recommended in cases in which the disease was at one time inflammatory, but the operation is of doubtful benefit. Sooner or later in all cases of interstitial nephritis the heart tends to fail, and cardiac tonics such as digitalis or strophanthus are necessary, while strychnin is often of great service.

SPECIFIC INFLAMMATIONS OF THE KIDNEY.

The inflammations which occur in typhoid, cholera, &c., are, properly speaking, specific forms. They have already been described. Of the chronic forms, the most important are *syphilis* and *tubercle*. The former has already been noticed. It produces either the parenchymatous (chiefly chronic) or the interstitial nephritis. The latter (renal tuberculosis) occurs in two forms, viz., the miliary and the caseous varieties. The former is never primary, but occurs as an incident in general miliary tuberculosis, or more frequently as a late manifestation of serious tubercular disease elsewhere, as in the lung, and hence is of no clinical importance. The latter is a chronic process: it may begin as a primary disease in the kidney, bladder, epididymis, or Fallopian tubes. In whichever place it begins, it tends to spread to other parts of the genito-urinary tract, and is thus a descending (more common) or ascending genito-urinary tuberculosis. It is more common in males than in females, occurring particularly in young adult age. The appearance presented by the kidneys depends on the stage of the disease. Caseous masses are found near the apices of the pyramids, which may break down and form small cavities communicating with the pelvis of the kidney. Their walls are formed of irregular caseous

material. They gradually increase in size, involving more and more of the pyramids, and subsequently of the cortex, until they reach and distend the thickened renal capsule. The kidneys (for though both are usually affected, one is more affected than the other) may thus become greatly enlarged and occupied by several large cavities whose purulent contents become altered to a putty or mortar-like consistence, around which may be many other similar, but smaller, foci or cavities scattered in the renal tissue. The pelvis is usually highly inflamed and ulcerated or lined with caseous material. The ureter is dilated and similarly affected. It is often blocked in its upper part by the caseous thickening and by plugs of caseous material coming from the kidney. This retards the escape of urine and may result in a hydro- or a pyo-nephrosis.

Symptoms.—A more or less constant pain in the back may be present, and spasms of pain like those of renal colic may occur, occasioned by the passage of caseous masses down the ureter. Frequent micturition is common, but the most frequent and important sign of all is the presence of pus in the urine. It may exist for years without much affecting the health. When pus appears insidiously in the urine without any assignable cause, tuberculous disease should always be suspected. The detection of tubercle bacilli in the urine is pathognomonic. The urine should be centrifugalised and the examination should be frequently repeated, for the bacilli occur in sparsely distributed clumps. Animal inoculation is of great value, and the tuberculin reaction may also be tried. Blood may be present, but it is usually slight in amount. The disease may recede and recovery follow, but usually it progresses and causes general disturbance, such as fever, wasting, and extension to other organs. The kidneys may be palpable, but seldom reach a large size, except when a co-incident

hydro- or pyo-nephrosis occurs. The disease attacks one kidney before the other, the extension generally taking a long time, so that excision of the affected kidney, particularly in women, may check the disease.

Treatment.—If one kidney alone is the site of tubercular disease, its removal should certainly be entertained, but it is necessary to assure oneself that the other kidney is healthy, and the examination of the urine obtained from each of the two kidneys by means of a Luy's separator is of great importance. Tubercular involvement of the bladder should contra-indicate operation, and in such cases urotropine might be tried, and also the usual treatment for tuberculosis, consisting of cod-liver oil, tonics, and, in certain instances, an open-air life.

PYELITIS.

Etiology.—Inflammation of the pelvis of the kidney may spread downwards from the kidney or upwards from the bladder, *i.e.*, it may follow a nephritis (when it is usually called pyelonephritis) or a cystitis, and in the latter case it is usually bilateral. But it may arise without any noticeable inflammation of either the kidney or bladder, as in certain infectious fevers, in anæmias and other debilitated conditions of the body generally. Cases following cold and over-exertion may be classed among the latter. In all cases it is the result of bacterial action, *viz.*, of either one or more of the organisms of ordinary inflammation, such as the bacillus coli, the staphylococcus, or of a special germ, such as tubercle. The ova of certain worms are said to cause it; but, contrary to the old time belief, a calculus is not a cause. It is possible for a calculus to so injure the mucous membrane of the pelvis that the latter falls an easy prey to some accompanying or following germ, but in most cases a pelvic calculus is a sequel to pyelitis and not its cause.

Morbid Anatomy.—The mucous membrane of the pelvis is swollen, of a greyish colour, often showing hæmorrhagic points, and some roughness. Occasionally a greyish false membrane is formed. The urine in the pelvis is cloudy, and owing to its collection therein there is a varying degree of dilatation of the calyces and flattening of the apices of the pyramids. The urine may become purulent (*pyonephrosis*). The inflammatory or suppurative process may extend into the kidney (*pyelonephritis*). Abscesses of varying size may extend into the kidney substance from the pelvis or appear discontinuously throughout the kidneys (surgical kidneys). If the liquid parts of the pus be absorbed, the abscess contents assume the putty or mortar-like appearance much more frequently seen in chronic renal tuberculosis.

Symptoms.—In the early stages the symptoms may be slight or absent. Pain and tenderness in the loins come on early. Frequent micturition is a prominent feature. The urine is *acid*, and at first shows merely large numbers of fusiform and tailed cells. Pus generally appears sooner or later, and may reach a considerable amount. It may appear intermittently, particularly when only one kidney is involved. Albumin is usually more abundant than is accounted for by the amount of pus. Blood is usually absent except in calculus or tubercular cases. Moderate fever (101° or 102°) is usually present, but it may rise higher, particularly when rigors occur. A definite swelling may be felt in the lumbar region, particularly in cases of pyonephrosis.

Diagnosis.—The passing of a large quantity of acid urine (which frequently contains pus), pain and tenderness in the loins, and moderate fever are the chief signs.

Prognosis.—Ascending cases have generally a grave outlook, but others, excluding those arising in fevers or in

severely debilitated persons, often run a favourable course and subside in a few weeks.

Treatment.—In an acute case rest in bed and the application of poultices or fomentations to the loins are the first indications. A hot bath is a convenient and ready method of applying counter-irritation as well as inducing diaphoresis. The patient should be encouraged to drink freely of barley water and other warm diluent drinks.

The next question is the desirability of treating the patient by means of urotropine (5 to 10 grains) or similar agents, which are, although possibly in very slight degree, urinary antiseptics. Pus, if present, may be diminished by the use of astringents such as the mineral acids or the tincture of the perchloride of iron, while certain volatile oils, such as sandal-wood oil or oil of turpentine, if given in small doses, often prove beneficial. In all cases the patient's general health should be well maintained and tonics administered. Sometimes operative interference is the only radical measure likely to result in cure, but neither nephrotomy or nephrectomy should be entertained unless the surgeon is convinced that the other kidney is capable of adequately carrying on its functions.

HYDRONEPHROSIS.

Etiology.—It may be congenital or acquired. In the former it is due to some obstruction in the ureter or urethra and is usually bilateral. In the latter it may be caused by anything capable of obstructing the free flow of urine down the ureter, such as a calculus, a plug of inflammatory exudate or pus, of cancerous or of tuberculous tissue blocking the lumen, or by kinking of the ureter in movable kidney, or by the pressure upon it from without by an abnormal artery, or by a tumor (particularly ovarian or uterine cancer or fibroids), or by peritoneal adhesions. The hydronephrosis may be transitory, permanent, or inter-

mittent when produced by any of these causes. It is frequently intermittent in movable kidney. The greatest dilatation is apt to arise when the obstruction is intermittent. Total obstruction may lead to atrophy of the kidney without any hydronephrosis. The obstruction may be below the ureter, as in cancer of the bladder, enlarged prostate, or urethral stricture.

Morbid Anatomy.—The pelvis is gradually distended and the apices of the renal pyramids flattened. This flattening and compression involves the whole pyramids and intervening renal tissue in time, so that the whole renal tissue becomes reduced to a mere shell not more than one-eighth of an inch thick in extreme cases. It forms the sac of the hydronephrosis. On the inner surface of the sac the walls of the calyces can be traced as septa (in which the renal blood-vessels lie) between the loculi of the hydronephrosis. The fluid is thin and yellowish in colour. When of old standing, it contains only traces of urea and other nitrogenous substances and urinary salts. It may contain albumin or pus, or even blood, which is occasionally present in large quantity, and may even form a large thrombus. Hypertrophy of the left side of the heart is usually seen.

Symptoms.—Congenital cases, when bilateral, usually prove fatal within a few days, when unilateral there are usually no symptoms until a tumor makes its appearance in the loin. In adult life, when caused by pressure, &c., the same thing usually occurs. When, however, the obstruction is complete and of sudden onset, there is pain in the back, nausea and vomiting, followed by a tumor in the loins, which is easily recognised as of renal origin when of moderate size, but may be confused with ovarian or other tumors when of large size. The tumor may suddenly disappear with the passage of a large quantity

of urine. This may occur again and again in the course of several years.

Diagnosis.—The history of the case and the presence of a renal tumor are often sufficient. In doubtful cases the sac may be aspirated and the fluid tested for urea, uric acid, urinary salts and cells.

Prognosis.—It depends on whether the condition is unilateral or bilateral and on the cause. When it follows upon calculus, enlarged prostate, or movable kidney, the outlook is favourable, when it arises in cancer or other tumor growth it is naturally grave. Uræmia may supervene in bilateral cases, and in all cases where the distension is great, the kidney is more or less permanently damaged.

Treatment.—Many cases require no treatment. An abdominal bandage has been recommended where the case is intermittent. Sometimes a hugely distended sac requires aspiration, free drainage, or may even necessitate the removal of the kidney altogether. Where puncture is attempted it should be carried out in the space between the last rib and the crest of the ilium.

CYSTIC DISEASE OF THE KIDNEYS.

Etiology.—There are two forms of general cystic disease of the kidneys—the congenital and the adult. Their etiology is obscure, the most generally accepted view being that they are of the nature of a new growth—a form of adenoma due to proliferation from the existing tubules. This view is supported by the frequent co-existence of cysts in the liver, pancreas and other organs. Other two views of their origin, however, are held by many: the one attribut-

ing the cysts to obstruction of the tubules, the other to some error in development, some mixing of the pre-renal tubules and consequent persistence of portions of the Wolffian body.

Morbid Anatomy.—The appearances are much the same in both varieties. Both kidneys are usually affected, though one more so than the other. The increase in size is often considerable. In congenital cases it may interfere with birth. In adult cases the kidneys may be three or four times their usual size. The general kidney shape is usually preserved, though the surface is altered by a multitude of projecting cysts. On section, the kidney substance is everywhere riddled by cysts with watery or jelly-like or mucoid contents of a clear yellow or various shades of red, brown or even black colour. The section presents a strikingly variegated appearance owing to the many shades of colour. The colouration is mostly due to blood and variously altered blood-pigment. Hæmorrhage appears to take place at some time or other into most of the cysts. The clear and watery cysts contain mere traces of urea and no urinary salts. In advanced cases little or no kidney substance may be seen between the cysts, but it exists often in surprising quantities when examined microscopically. It may be but slightly altered from the normal, but more usually it shows varying degrees of interstitial nephritis. The cysts themselves may be lined by a flattened or columnar epithelium.

There are other varieties of cysts than the general cystic form described above, of which the most notable are the multiple obstruction cysts, varying in number, generally small in size, and more numerous in cortex than medulla, met with in chronic interstitial nephritis or granular kidney, and the large single cyst (sometimes two or three) seen in normal kidneys. Some regard this large cyst as congenital, but most consider it as obstructive in origin.

Symptoms.—In many cases there are no symptoms at all, or only towards the end, when uræmia develops suddenly. In others the patient shows the polyuria, anæmia, &c., characteristic of chronic interstitial nephritis. Recurrent hæmaturia is frequently present. In later stages enlarged tumors may be felt in the region of the kidney behind the colon.

Treatment.—The treatment is usually entirely confined to the relief of symptoms. It is rarely possible to carry out any surgical procedure likely to prove of benefit to the patient.

TUMORS OF THE KIDNEY.

Morbid Anatomy.—Simple tumors, such as lipoma, fibroma, angioma and adenoma, occur in the kidney, but rarely grow to any size. A simple adenoma may occasionally reach the size of a walnut, but even then it does not cause trouble during life. The adenomata which arise from the collecting tubules are apt to become cystic and papilliferous and reach a considerable size. Malignant tumors, on the other hand, grow to a large size and rapidly cause death. Sarcoma is more frequent than cancer. It is mostly either congenital or makes its appearance in the first months of life, and owing to its comparative frequency is one of the most important of the tumors of childhood. They form large, soft, whitish, vascular tumors, restricted within the kidney capsule at first but usually extending beyond it after a time and constituting large irregular and nodular masses in which the normal shape of the kidney may be lost. Microscopically, they show round or spindle cells, or both, and many of them, particularly the smaller ones, contain large spindle-shaped muscle cells with transverse striations. When these muscle cells are present the tumor is called a rhabdomyosarcoma or a

rhabdomyoma. All the varieties are very vascular and are liable to grow into the pelvis and invade the renal vein, hence hæmaturia is a very common occurrence. The congenital varieties are the most rapid growers, and generally prove quickly fatal. The acquired varieties, on the other hand, may grow slowly, particularly when they arise later in life, and may not give rise to metastases for a long time. Cancers of the kidney originate in the renal epithelium and are comparatively rare. The great majority of epithelial tumors found in the kidney have not originated in the renal epithelium at all, but in tissue elements originally derived from the suprarenal body and included within the kidney substance during development. They are called *hypernephromata*, or the "aberrant rests" of Grawitz. Kelly states that most of the primary cancers and alveolar sarcomas of the kidney are hypernephromata. Whether this be so or not, hypernephromata are common and form pea or cherry-like or larger-sized well-defined subcapsular growths. The larger cancers form circumscribed nodular growths, invading the kidney substance or entirely replacing it, but remaining restricted within the capsule, for even when the size is three times or more that of the normal kidney, the general outline of the kidney is preserved, the capsule is intact, though thickened, and there is no infiltration of the perirenal tissues; hence the tumor can be easily shelled out. On section of such a tumor little or no kidney tissue may be detected, the whitish nodular cancer having everywhere destroyed it. Hæmorrhages into the substance of the tumor are frequent, and invasion of the renal pelvis and the renal vein by the tumor growth is also common. Renal tumors are nearly always unilateral, though a very few cases of bilateral cancer have been recorded.

Symptoms.—Pain, hæmaturia and the presence of a tumor are the three leading indications of renal cancer.

The pain is uncertain. When present, it is usually of a dull character and situated in the loin. Attacks of acute pain like that of renal colic are not uncommon. They are caused by the passage of blood clot or of fragments of the growth. Hæmaturia is absent in about one-half of the cases throughout the whole course of the disease. It is usually intermittent, lasting for a few days at a time, but it may be persistent and occur daily for months, or even years. It varies in amount and may be liquid or form clots of the renal pelvis or ureter. The tumor is detectable comparatively early after the symptoms begin to appear in almost all cases. It occupies the lumbar region and may be movable or fixed. The colon can be felt to pass in front of the tumor, whereas it lies behind splenic tumors, which, moreover, generally show the characteristic splenic notch. In later stages emaciation may become prominent, and there may be distension of the veins of the abdominal wall and œdema of the legs from occlusion of the inferior vena cava, either by the pressure of the large tumor or by an invasion of the renal vein and vena cava by cancerous growth or by thrombosis.

Prognosis.—Death usually follows in about a year after the symptoms begin, though cancer occasionally lasts for years. Death is certain to follow, however, unless nephrectomy be performed. This has been fairly successful in cases of adrenal rests, less so in sarcomas and cancers.

Treatment.—Surgical treatment is advisable in a proportion of the cases in which the tumor is primary and apparently limited to one kidney. Usually it is only possible to relieve symptoms, and one of the chief of these—pain—demands the use of sedatives such as opium, and the patient should be kept in bed. Blood, when passed in excess in the urine, may require the internal administration of astringents. Sometimes soothing external applica-

tions prove of comfort to the patient. Simple tumors do not call for any treatment.

PERINEPHRIC ABSCESS.

Etiology.—Suppuration around the kidney usually has its origin in the kidney itself, particularly in its pelvis. Suppurative pyelitis may lead to ulceration of the mucous membrane of the pelvis, or to burrowing of the pus into the perinephric connective tissue. Similarly, a suppurative inflammation of the kidney or ureter may invade the perirenal tissue, and so also may inflammation of the appendix, colon, vertebral column (caries), pleura or any other neighbouring structure. Occasionally there is no preceding inflammation, but merely a condition of lessened resistance, for the perinephric abscess may follow upon blows or other injuries to the loins or upon some of the specific fevers, particularly in children.

Morbid Anatomy.—The abscess is usually large and the pus may surround the kidney or be restricted chiefly to its posterior or anterior surface. It may burrow in various directions, particularly downwards towards the groin, or discharge itself into the colon, peritoneum or pleura.

Symptoms.—Pain in the loin and the position of the leg are the most marked features of the disease. The pain is present in most cases, though it is absent in some cases which begin insidiously. It is sometimes very severe. It is felt in the loin and frequently radiates downwards to the hip joint and thigh of the same side. The thigh on the affected side is flexed at the hip so as to relax the psoas muscle, as in psoas abscess. In walking, the spine is kept immobile, the thigh flexed, and the body weight thrown as

much as possible upon the opposite side. On bi-manual examination a tumor with distinct fluctuation can usually be made out. Fever and rigors may occur and pus may appear in the urine.

Treatment.—The treatment required is purely surgical.

Section 8.

DISEASES OF THE NERVOUS SYSTEM.

INTRODUCTION.

The morbid changes arising in the nervous system are simple enough in themselves, being similar to those found in other parts of the body. It is not they which make the study of nervous diseases a complicated one, but the complexity of anatomical arrangement and the great specialisation of function in its different parts which do so. The student who starts upon a study of the diseases of the nervous system already equipped with a competent knowledge of the anatomy and physiology of the brain and spinal cord finds an easy task before him. It is advisable, in most cases, to freshen up this knowledge. A few of its most important facts may therefore be usefully referred to here, but the student is strongly advised to read afresh the anatomy and physiology of the nervous system.

THE NEURONE.—The simplest conception of the central nervous system regards it as being made up of an immense number of nervous units, or neurones, separated and supported by a connective tissue or neuroglia. Each neurone consists of a cell body and processes. The cell body possesses protoplasm, a nucleus, and nucleolus. The nucleus is the most important part of the cell. The proto-

plasm consists of two kinds of substance ; the one—achromatic—sometimes called the hyaloplasm, does not stain with basic aniline dyes like methylene blue, the other—chromatic—takes the form of granular particles of protoplasm, varying much in size or shape, which stain deeply with methylene blue. These particles are known as “tigroid,” or Nissl’s, bodies. The achromatic substance contains bundles of fine fibrils which cross one another on their way to the processes of the cell body, and it is in the meshes thus formed that the Nissl bodies lie. These fibrils pass through the cell body from process to process, and the majority of them do not anastomose in their transit through the cell. The cell processes are of two kinds. All the processes, or their majority, except one, divide and subdivide shortly after leaving the cell, until each ends in an arborescence of fine twigs which surround the cell body. These processes are known as dendrites. Most of them show little lateral buds, known as gemmules or thorns, which increase their conducting surface. The exceptional process is of a different kind. It is the axis-cylinder process, and is known as the neuraxon or axon. There is only one for each cell body. It generally runs a long course. After passing for a variable distance from the cell body, it gives off at right angles a collateral branch, and continues to give off similar collaterals throughout its course, until it terminates in arborisations in an end organ, such as a muscle, or around other nerve-cell bodies. Its collaterals terminate in the same way. These arborisations intertwine with those of the cell bodies they surround, but they are believed not to anastomose with them. This relationship is technically known as a *synapse*. They are contiguous, not continuous ; but though not anatomically connected, messages can pass with ease from the one to the other. The dendrites are afferent, *i.e.*, conduct impulses towards the cell bodies, and the neuraxones are efferent, *i.e.*, conduct them away from the cell bodies. Both the dendrites and the axones con-

tain bundles of fibrillæ which enter the cell body, passing through it without anastomosing therein to any extent, and emerge by entering other processes—either dendrites or axones. Nissl's granules extend for some distance into the dendrites, but not into the axones. The main constituent of the Nissl granules is a nucleo-proteid. Their actual relationship to the protoplasm and to the nucleus is still doubtful. They are constantly present in healthy cells after staining with methylene blue, and it is therefore probable that they are connected with, and in some measure express, the vital interaction between the highly phosphorised nucleus and the surrounding protoplasm. Their disintegration and diffusion throughout the protoplasm or disappearance is known as *chromatolysis*. It is seen in various abnormal states, particularly during the action of toxins. It begins at the periphery of the cell and in the dendrites, and in advanced cases extends throughout the cell. It is always a retrograde change, but does not necessarily mean the death of the cell. So long as the vitality of the nucleus remains unimpaired, the cell may recover completely.

The axone, soon after it leaves the cell, acquires a sheath—the medullary or white sheath of Schwann. No other sheath is present within the brain or cord, but in the peripheral nerves there is an outer sheath or neurilemma, composed of epithelial cells whose nuclei are seen about the middle of the internodes, *i.e.*, the parts of the nerve-fibre lying between the nodes of Ranvier, *viz.*, the regular interruptions which occur in the continuity of the white sheath.

Each cell body exerts a trophic influence over all its processes. If the axone be cut across, the distal end rapidly degenerates as far as its terminal arborisations in a muscle or around another cell. When this occurs in a peripheral nerve, regeneration quickly follows. This consists in a formation of a new axis-cylinder process from

the central end, aided, it may be, by the distal end. The epithelial cells of the neurilemma of this distal end proliferate, become phagocytic, and later elongate and join together. Some authors assert that they form new nerve-fibres in this way, but it is much more probable that they only form a new sheath into which the axis-cylinder sprouting from the central end ultimately grows. No other change in the central end or in the nerve-cell body in the spinal cord occurs. On the other hand, when regeneration does not occur, as it never does in the central nervous system, the cell body and its process slowly undergo retrograde changes. This *reaction a distance* consists in a chromatolysis, an initial swelling followed by an atrophy of the cell body, a diminution in the grey processes, and a dislocation of the nucleus.

The neurones are collected together into systems within the brain and cord, their cell bodies within the grey matter, and their axones within the white matter. The most important and best known of these systems is that in which the motor neurones are collected, and it is known as the *efferent or motor system*, the next in importance being that containing the sensory neurones—the *afferent or sensory system*.

THE MOTOR SYSTEM.

The ascending frontal convolution contains all the cell bodies of the neurones which are concerned in muscular movement. In man and the higher apes they are restricted to this convolution, although in the lower apes some of them reside also in the ascending parietal convolution. These cell bodies in certain combinations constitute the motor centres which preside over the movements of the different parts of the body. These centres are widely spread throughout the ascending frontal convolution, those for the head being lowest down, those for the arm

being next higher, those for the trunk still higher, and those for the leg being highest of all. This frontal convolution has indeed been aptly likened to the spinal cord upside down. The centres for the head and arm occupy a much larger area than those for the trunk and leg. Every muscular movement, even the simplest, involves the activity of many neurones. They must combine in a definite way to produce any specific movement, and the more complex the movement the greater is the number of neurones required in combination. The greater complexity of the movements executed by the head and arm, compared with the trunk and leg, therefore explains the larger area occupied by their motor centres. The centres for the face and jaws take up the largest part of the head region, those for the tongue occupying its lowest part, while those of the neck comprise a comparatively small area in its highest part, where it borders upon the arm region. Immediately in front of the centres for the tongue on the left side is the centre for motor speech, occupying the posterior part of the third frontal, or Broca's, convolution: in left-handed people it is situated on the right side of the brain. The motor centres of the left side of the brain govern the muscles of the right side of the body, and *vice versa*, the neurones mostly crossing in the pyramids of the medulla. This is seen by tracing the course of the neurones downwards from the cell bodies in the grey matter of the ascending frontal convolution. Their axis-cylinder processes radiate into the white matter of the corona radiata, whence they converge to be gathered together into a narrow and compact bundle in the pyramidal tract in the internal capsule, occupying its knee and the anterior two-thirds of its posterior limb. When the axones start from the cortex those for the leg are highest up and those of the head lowest, but by the time they reach the internal capsule those of the head have become highest, occupying the knee part of the capsule, and those of the leg lowest,

occupying the mid third of its posterior limb, whilst those of the arm lie between, in the anterior third. After the pyramidal tract emerges from the internal capsule it passes through the crus, where it occupies the middle part (about three-fifths) of the crura, the axones of the lips and tongue being nearest the middle line. Some of the axones here leave the pyramidal tract, cross the middle line, and end in arborisations around the ganglion cells in the nucleus of the third nerve on the opposite side. The pyramidal tract then passes through the pons and enters the pyramid of the medulla. In its course through these structures axones continue to leave it and cross the middle line, to end in arborisations around the ganglion cells of the nuclei of each of the motor cranial nerves. When the last of these has been given off, the pyramidal tract has reached the lower end of the medulla, where the great majority of its axones cross the middle line, decussating with those from the opposite pyramid, and descends in a tract in the lateral part of the cord, called, for this reason, the crossed pyramidal tract. The small number of axones which do not cross here descend the cord in the antero-median tract, called, for this reason, the direct pyramidal tract. Throughout the whole of their course in the cord, as in the crus, pons and medulla, axones continue to leave the tracts, to end in arborisations around the ganglion cells of the anterior horn, on the same side in the case of the crossed, and on the opposite side in the case of the direct tracts. Schäfer holds that the pyramidal fibres really terminate around cells at the base of the posterior horn, whence they are transmitted by association fibres to the cells of the anterior horn, the posterior horn cells acting as intermediate stations. Both tracts continue to get smaller from above downwards, the direct ending about the middle of the thoracic region of the cord, and the crossed extending throughout its length, although it is reduced to a very small size at the end of the cord. At the

lower part it extends to the margin of the cord, but higher up the direct cerebellar tract is interposed between it and the margin. These motor neurones, which start in the cerebral cortex and end around the cells of the cranial nuclei or of the anterior horn, constitute the upper part of the motor system, and some of them are the longest in the body. A fresh set of neurones starts in the ganglion cells of the motor nuclei of the cranial nerves and of the anterior horn of the cord. Their axis-cylinder processes leave the cell bodies and pass into the peripheral nerves. The spinal ones leave the cord as several bundles, which unite together to form the ventral or motor root of a spinal nerve. Each spinal nerve is formed by the union of a motor and a dorsal or sensory nerve root, and that part of the cord from which they arise is known as a segment. Each segment gives off its corresponding nerves on each side, and while these correspond in number to the vertebræ in the cervical region, they do not do so lower down, because the bony canal grows faster than the spinal cord. Hence in the lower part of the cord the segments lie considerably higher than the vertebral bodies corresponding to them in number and name. The axones going into any spinal nerve do not all arise from the corresponding segment, some being derived from cells situated in other segments. The motor fibres of each spinal nerve end in arborisations within a muscle, and its sensory fibres in arborisations in a tendon or in the skin. This second or lower set of motor neurones thus extends from the basal cranial nuclei in the pons and medulla or from the cord to the muscles. It is direct and not crossed. Both sets of neurones are necessary for every muscular movement. Such a movement starts in some of the cell bodies of the upper set of neurones, and is transmitted by their axones through the brain and cord to the cell bodies of the lower set, whence it is transmitted to the muscles. A lesion may

affect either the upper or the lower set of neurones in any part of its course. Irritative lesions induce activity of the neurones, whilst destructive lesions paralyse them. Irritative lesions, *e.g.*, tumors, occur oftenest in the cerebral cortex. They cause spasmodic movements, or Jacksonian epileptic attacks, involving particular groups of muscles in the arm, leg or face ascending to the centres involved. A Jacksonian convulsion begins in a muscle, or group of muscles, and extends to neighbouring muscles in a definite order. Destructive lesions occur in any part of the motor system. *In the upper set* the lesion may be in the cerebral cortex and cause paralysis of motion in the face, arm or leg on the opposite side according to its size and to its position. This produces the condition known as *cerebral monoplegia*. It is rarely confined to a single group of muscles. If such a lesion be sufficiently extensive, which it rarely is, it may involve more than one area, *e.g.*, both arm and leg centres, and thus cause paralysis of motion of both arm and leg on the opposite side, a condition known as *hemiplegia*. On the other hand, if a destructive lesion of any size occur in the basal ganglia or internal capsule, and this is common, hemiplegia of the opposite side follows, since the motor fibres are gathered together into a small and compact bundle in this situation. This is also the case lower down in the pons and medulla, but since the motor fibres of both sides lie near each other, on either side of the middle line, both may be affected, producing paralysis on both sides of the body. Below the decussation of the pyramids, *i.e.*, when the lesion is in the crossed pyramidal tract, the paralysis is on the same side of the body. In all these lesions of the upper set of motor neurones the motor fibres lying below the seat of lesion will gradually degenerate, since they are cut off from their trophic cell bodies. Rigidity of the paralysed muscles follows, but the muscles do not waste, nor yet

do they show the reaction of degeneration. Further exaggeration of tendon reflexes soon makes its appearance, owing to the loss of voluntary control over the reflex arcs. *In the lower set of motor neurones* the lesion may be in the cell bodies of the anterior horn, as in infantile paralysis; in the ventral nerve roots, as in spinal meningitis; or in the peripheral nerves as in peripheral neuritis. The paralysis involves single muscles or groups of muscles, as contrasted with whole sections of the body in lesions of the upper set of motor neurones. The affected muscles atrophy. They show the reaction of degeneration. This consists in their responding in a slow and sluggish way to the galvanic current, in their contracting often in response to a weaker current, and in a reversal of the influence of the negative and positive poles. With the galvanic current applied to a normal muscle, on closing the circuit the negative pole or cathode gives an earlier and stronger contraction than the positive pole or anode. This is expressed thus: the cathode closing contraction is greater than the anode closing contraction $C.Cl.C. > An.Cl.C.$ In the reaction of degeneration the $An.Cl.C. > C.Cl.C.$ The degenerated nerve is not stimulated by either the galvanic or faradic current. These are the changes when the reaction of degeneration is complete. They develop in from seven to ten days, and are of grave significance. When it is incomplete the galvanic and faradic excitability of the nerve is lessened only and not lost, while the muscle response to galvanic stimulation is slower than normal, the polar reaction being equal $An.Cl.C. = C.Cl.C.$ Rapid recovery is frequent in such cases.

Abolition of both the superficial and deep reflexes is a further sign of a lesion of the lower motor neurone since it is the efferent limb of the reflex arc. The superficial reflexes are true reflexes. They are obtained by gentle stimulation of the skin. The responding

muscles lying beneath the part of the skin touched are those which contract, though occasionally others at a distance do so also, *e.g.*, a prick near the knee causes reflex flexion of the hip. The chief superficial reflexes (with their corresponding segments of the cord) are:—(1) *plantar reflex*—flexion of the toes and withdrawal of the feet when the soles are tickled (first sacral to second sacral segments); (2) *gluteal reflex*—contraction of the gluteal muscles when the skin of the buttock is stimulated (fourth lumbar to fifth lumbar segments); (3) *cremasteric reflex*—contraction of the cremaster with drawing up of the testicle when the skin on the inner side of the thigh is gently stroked (first lumbar to second lumbar segments); (4) *abdominal reflex*—contraction of the abdominal muscles when the skin of the side of the abdomen is stroked: it is marked at the epigastrium, known as the epigastric reflex (ninth dorsal to twelfth dorsal segments); (5) *scapular reflex*—contraction of the scapular muscles by drawing the finger over the skin of the shoulder (fifth cervical to first dorsal segments). In the head the two most important reflexes are:—(1) *conjunctival reflex*—shutting the eyelids when the front of the eyeball is touched; (2) *pupil reflex*—contraction of the pupil when the eye is exposed to light, and its dilatation when the skin of the neck is stimulated. The deep reflexes are:—(1) *the patellar tendon reflex or knee jerk*—the sudden projection of the foot forward when the patellar tendon is sharply tapped after being put slightly on the stretch by crossing the knee (second lumbar to fourth lumbar segments); (2) *ankle clonus*—a clonic series of contractions of the calf muscles when the hand is suddenly pressed against the sole of the foot, thus putting the calf muscles on the stretch (first sacral to second sacral segments); (3) *triceps-jerk*—jerking of the arm when, with the arm flexed, the triceps tendon is sharply tapped

(sixth cervical to seventh cervical segments). There are other tendon reflexes of less importance which need not be described. These so-called tendon reflexes are not true reflexes, for the time intervening between the tap and the response is too short to permit of a message travelling upwards to the cord and downwards again to the muscle. It must be due to the direct stimulation of the muscles by the sudden stretching of their tendons. When the reflex arc is intact the muscles are easily put into a state of tonus or myotatic irritability by slightly stretching them. In other words, they are ready to contract on the slightest provocation. When any part of the reflex arc is injured the healthy tone of the muscle is abolished, and hence the irritable condition is not produced when it is suddenly stretched, and the deep reflexes disappear.

THE SENSORY SYSTEM.

The course of the sensory fibres is much more complex than that of the motor. It has not yet been completely worked out. It may be conveniently arranged in three groups of neurones; the first extending from the skin to the nucleus gracilis or cuneatus, the second from these nuclei to the optic thalamus, and the third from it to the sensory cells in the cortex.

The first group of sensory neurones.—Each ganglion, the cell body of the neurone, on the posterior root of a spinal nerve gives off one process, which divides into a lower and an upper branch. The lower runs to the periphery of the body, where it terminates in arborisations in the skin and in tendon, from which it conducts impulses upwards. It represents the dendritic process of the ganglion cell. The upper branch (the axis-cylinder process) enters the spinal cord, where it bifurcates into an upper and lower fibre; the latter runs downwards in the posterior comma tract of the

cord for a short distance, and terminates in an arborisation around one of the cells of the posterior horn, from which a new axis-cylinder process arises and arborises around one of the cells of the anterior horn. This descending branch gives off a number of collaterals which terminate in a similar way. This constitutes the reflex arc. The former, *i.e.*, the upper branch, is the main division. It travels up the posterior columns of the cord, giving off collaterals at various levels, and terminates by arborising around a cell of the nucleus gracilis or nucleus cuneatus of the medulla oblongata. Many of the main branches do not reach so high, but terminate in the same way as the collaterals. None of these fibres cross the middle line. Some of the collaterals arborise directly around cells of the anterior cornu, others also do so indirectly by first arborising around cells in the posterior cornu, which act as intermediate cell stations, and yet others arborise around cells of Clarke's column. Some of the axones given off by cells of the posterior horn form the ventral cerebellar or antero-lateral tract of Gowers. Some fibres decussate, for example, those of common and of tactile sensation and of temperature, whilst those carrying muscular sense pass up on the same side of the cord.

The second group of sensory neurones begins in the ganglion cells of (1) the nucleus gracilis and cuneatus, (2) Clarke's vesicular column, and (3) cells of the posterior horn. The axones of (1) cross the middle line as internal arcuate fibres, and become longitudinal in the fillet, to terminate around cells in the ventro-lateral part of the optic thalamus. The axones of (2) run upwards in the direct or dorsal cerebellar tract, then through the restiform body or inferior peduncle of the cerebellum, to terminate around cells in the vermis or middle lobe of the cerebellum. The axones of (3) run upwards in the ventral cerebellar tract, then through the superior peduncles of the cerebellum, to terminate also in the vermis.

The third group of sensory neurones begins in ganglion cells of the optic thalamus. Their axones pass to cells in the cerebral cortex, behind the Rolandic fissure, from which association fibres arborise around the motor cells of the ascending frontal convolution. The axones from the cells of the vermis around which the sensory arborisations terminate are supposed to pass down the cord to end around the cells of the anterior horns.

The grey matter is continuous from the spinal cord to the optic thalamus, and it is believed that certain afferent impulses, such as those of pain, may travel directly upwards by its means. The anatomical arrangement of the sensory fibres becomes more complicated as the various tracts pass into the brain, and it is hardly yet well enough known to be of much clinical value, except in the cord, or rather in the course occupied by the first and second groups of sensory neurones. Irritative lesions cause abnormal sensations, e.g., pain, paræsthesia, formication, a sense of cold or of constriction. Destructive lesions cause loss of sensation below the seat of lesion. Total transverse section of the cord causes complete sensory and motor paralysis below the lesion, abolition of the deep reflexes, and rapid wasting in the paralysed muscles. Hemisection or a complete unilateral lesion of the cord also causes sensory, motor and reflex disturbances, but of a different kind. The combination of symptoms in such cases is often called *Brown-Séquard's* paralysis, after its discoverer. The motor and reflex symptoms are confined to the side of the lesion, but the sensory symptoms are not. *The motor symptoms.*—The muscles whose centres of innervation lie at the site of the lesion are flaccid and paralysed. They atrophy and give the reaction of degeneration. The muscles whose nerve centres lie below this level are also paralysed, but they are not flaccid, nor do they atrophy. They become spastic and retain their normal electrical reactions. *The reflex symptoms.*—The deep reflexes, after

being lessened, are increased. Babinski's sign is positive. *The sensory symptoms.*—On the same side as the lesion, and at its level, there is a zone of complete anæsthesia. Immediately above the lesion there is often a zone of hyperæsthesia, and below the lesion there is usually hyperæsthesia, impairment of muscular sense, and increase of the surface temperature. On the *opposite* side from the lesion, and nearly up to its level, there is a greater or less degree of loss of the tactile sense and complete loss of perception of pain and temperature. A perfect example of the Brown-Séquard's paralysis is rarely met with in disease in man, but modifications of it are seen in syphilis and tumors of the cord, and in intra-spinal hæmorrhages.

DISEASES OF THE COVERINGS OF THE BRAIN AND OF THE CORD—THE MENINGES.

The coverings of the brain and of the cord are called the meninges. They are liable to hæmorrhage, inflammation, and tumors. Inflammation of the dura is called *pachymeningitis*; of the pia-arachnoid, *leptomeningitis*.

DURAL HÆMORRHAGE.

Hæmorrhage may occur on the outer or inner surfaces of the dura or into its substance. The former is the only frequent form. It is usually the result of a fracture of the skull, and the middle meningeal artery is its most frequent source; but it may come from a wounded sinus, especially the lateral sinus. Injury at childbirth, particularly in difficult labours, is also a common cause. The blood varies in amount and position, but it always collects between the dura and the bone, causing compression of the brain. The symptoms of compression usually come

on some little time after the injury, and tend to get worse. Complete or partial paralysis is one of its most important indications, particularly when it is accompanied by a slow, laboured pulse and respiration, and by insensibility. If death does not result, absorption and organisation occur in the blood, whilst the compressed brain cortex becomes softened and atrophied. Some of the infantile cerebral palsies or birth palsies arise in this way.

PACHYMENINGITIS.

The inflammation may involve the outer or the inner surface of the dura.

PACHYMENINGITIS EXTERNA.

The cerebral form is the result of syphilitic disease of the skull bones (most common), of middle ear disease, or of injury. A swollen, discoloured, and softened condition of the outer layers of the dura is produced, with (usually) the formation of pus between it and the bone. The amount of pus is rarely large and rarely diffused. Symptoms of compression may result, which usually improve if the pus dries up. In other cases the pus burrows through the dura, causing a pachymeningitis interna and a leptomeningitis. If the dural sinuses are involved, a septic thrombosis follows. The cerebral form occasionally runs a chronic course, resulting in much fibrous thickening of the outer surface of the dura. The spinal form is rare in comparison with the cerebral variety. It is the result of inflammation of the bodies of the vertebræ, *e.g.*, tubercular disease of the spine, or of the surrounding soft tissues, *e.g.*, a bed sore.

PACHYMENINGITIS INTERNA.

A purulent form is met with as a sequel of external inflammation, and a tubercular form as an extension from

without, in the cord, or as an extension from within, *i.e.*, from a tubercular leptomeningitis, in the brain, but both are rare. A hæmorrhagic form, on the other hand, is comparatively common and merits special description.

PACHYMENINGITIS INTERNA HÆMORRHAGICA.

THE CEREBRAL VARIETY.—**Causes.**—It occurs most frequently in certain forms of insanity, particularly chronic senile dementia; but it also occurs in syphilis and all wasting diseases associated with marked atrophy of the cerebral convolutions, as in some cases of phthisis and anæmia.

Morbid Anatomy.—The inner surface of the dura is thickened. The thickness may be slight or considerable. It is red in colour, generally resembling blood clot, and is often laminated. Microscopically, it shows either a very vascular delicate connective tissue, layers of blood clot, or a combination of both, in which the clot lies next to the dura, hence the condition is sometimes called hæmatoma of the dura. It is probably inflammatory in origin, a very vascular fibrous tissue being thus formed, in which the blood-vessels, having very delicate walls, are prone to hæmorrhage, which may be so extensive as to destroy all traces of the first-formed fibrous tissue. This hæmorrhagic exudate is most frequently seen in the vertex, close to the falx cerebri, but may extend over the parietal parts of the brain or be seen over the frontal or basal regions.

Symptoms.—They may be entirely absent, particularly in very chronic cases, or there may be symptoms of compression.

THE SPINAL VARIETY.—It may occur alone or be combined with the cerebral form. Its causes and char-

acters are the same. A special form involves chiefly the cervical region, and is known under the name of pachymeningitis cervicalis hypertrophica. It is characterised by a fibrinous or fibrous (according to its duration) deposit on the inner surface of the dura, which may be so great as to increase the thickness of the dura to five or ten times its normal. The substance of the cord is compressed, and secondary degeneration is often seen in its tracts. The central canal may be dilated. This form of meningitis may be limited to a small part, or extend over a large part of the cervical enlargement. The nerve roots are generally compressed.

Symptoms.—The disease usually begins with intense neuralgic pains along the course of the arm nerves whose roots are involved. Great variability of these pains is a characteristic feature. Other sensory phenomena are also present, such as anæsthesia, hyperæsthesia, numbness, and tingling. Motor weakness and atrophy in the arm muscles follow, particularly in the flexors, producing a claw-like condition of the hand. When the secondary degenerations in the spinal tracts have developed, which generally occurs in time, sometimes two or three months or more after the onset of the disease, the lower extremities will show a condition of spastic paraplegia. The clinical picture of the disease and its chronic course are usually very characteristic, but at times there is difficulty in distinguishing it from syringomyelia, from amyotrophic lateral sclerosis, and most of all from tumors.

Treatment.—In traumatic cases, operative interference at the time of injury might be helpful. Later, pain may demand the administration of sedatives, and everything possible should be done to arrest the wasting of affected muscles. In cases in which improvement is commencing, massage and the careful use of electricity are indicated.

TUMORS OF THE DURA.

TUMORS OF CEREBRAL DURA.

Of the simple tumors, osteoma is the most frequent. It is mostly an extension of an exostosis of the skull, but separate bony growths may be found in the dura, particularly in the falx cerebri or in the tentorium. Fibroma and chondroma also occasionally occur. These simple tumors are comparatively rare and unimportant. Of malignant tumors, both primary and secondary sarcomata and secondary cancers occur. The secondary tumors may be very destructive. The primary sarcomata belong to the ordinary spindle-celled variety, or to special varieties, such as the psammoma or endothelioma. The ordinary sarcoma occurs either as a diffusely spread, or as a localised growth of the inner surface of the dura. The psammoma is always a localised growth, also from the inner surface. It is usually small and symptomless, although it has caused pressure upon the third or the fifth nerves. The endothelioma grows from any part of the dura, and may form large tumors invading both skull and brain.

The Symptoms of these dural tumors are generally those of compression. Muscular spasms, particularly of the Jacksonian type, are frequent.

Treatment.—Operative interference is sometimes most successful, and where there is sufficient inducement and definite localising symptoms, the skull should be trephined without delay.

TUMORS OF SPINAL DURA.

The tumors involving the dura mater of the spinal region may arise extra-durally in the cellular tissue, between it and the spinal column, or in the tissues outside the

spinal column, and invade the spinal canal through the intervertebral spaces. Lipoma is the most frequent of these extra-dural tumors. Myxoma and sarcoma are not infrequent. The last two, along with fibroma, also occur as intra-dural tumors arising either from the inner surface of the dura, or, as is more common, from the pia. Tumors are found at all levels of the cord, though most frequently in the lumbar and cauda equina regions. The symptoms of extra-dural tumors are referable for a time to the nerve roots. Radiating pains, anæsthesia, hyperæsthesia, girdle sensation, and muscular spasms (usually limited for a time to one side of the body) are the chief of these symptoms. Later, when the cord becomes compressed, paralysis gradually supervenes and gradually increases.

Treatment.—Laminectomy has been attempted in cases which are not of a syphilitic nature, and the results are encouraging. Simple tumors do not always call for interference, but where they do so the prognosis of the operation is favourable.

LEPTOMENINGITIS.

Leptomeningitis may be acute or chronic, primary or secondary. It is frequently distributed over the whole cerebro-spinal axis, though it may be limited to either the brain or cord, the latter more rarely.

ACUTE PRIMARY LEPTOMENINGITIS.

Three forms occur—a tubercular, a meningococcal, and a pneumococcal meningitis.

TUBERCULAR MENINGITIS.

This is caused by the tubercle bacillus. Most cases of tubercular meningitis come on in the course of obvious tubercular disease in some other part of the body, notably in the lungs. Many cases follow upon a more or less slumbering tubercular lesion, *e.g.*, a caseous bronchial gland, which gave rise to no symptoms during life. These are all to be classed under secondary meningitis. Do any really primary cases occur? It is very doubtful if they do, though theoretically there is no reason why the tubercle bacillus should not make its first attack upon the meninges, as it is believed to do upon the pleura. At the same time, it is a fact that in the vast majority of cases of tubercular meningitis a careful post-mortem examination discloses a pre-existing tubercular lesion in some other part of the body. It may be difficult or impossible to detect many of these lesions during life, and the tubercular meningitis appears to the clinician to be the primary disease. This is of such frequent occurrence that cases of tubercular meningitis form the majority of all cases of apparently primary meningitis, particularly in children, met with in practice.

Morbid Anatomy.—*In the brain.*—The blood-vessels of the pia may be dilated. There is effusion into the sub-arachnoid space, either serous, sero-fibrinous, fibrinous, or purulent (rare) in character. Its amount may be slight or considerable. It is generally most marked at the base, particularly around the pons and optic chiasma and over the perforated spaces, spreading into the Sylvian fissure, and often reaching the lateral surface of the hemispheres. In addition, there are minute grey granulations, so small as to be hardly visible to the naked eye, or as large as pin heads, scattered everywhere in the exudate, but particularly seen along the

lines of the blood-vessels. The soft membranes containing the exudate can be easily stripped from the brain, and if floated out in water over a black background the minute tubercles can be more distinctly seen. Under the microscope they show as cellular collections of typical structure, caseated in the centre, though giant cells are often absent. There are sometimes also minute pial hæmorrhages in the more acute cases. The cerebral convolutions are always more or less flattened, often greatly so, particularly in the base. The flattening is due to a distension of the lateral ventricles, and its degree is proportionate to the extent of the latter. The distension of the ventricles with a turbid fluid is so characteristic of the disease that it is frequently known under the synonym of acute hydrocephalus. The ependyma of the ventricles is often softened and rough, but this is believed to be merely a form of post-mortem maceration. The grey matter of the convolutions always shows some inflammation, *i.e.*, a varying degree of encephalitis. In certain cases of tubercular meningitis, one (sometimes more) large caseous tubercular mass, sometimes as large as a hen's egg, may be found imbedded in the superficial cerebral substance, but in touch with the pia in some part. It is generally formed by the confluence of several tubercular masses, and each may show a caseous centre and grey periphery, or the whole mass may be yellow and caseous, except a very thin part of its periphery, which is of a whitish-grey colour. It is most frequently seen in the cerebellum, but may occur in any part of the cerebral cortex. It usually causes no trouble unless it encroaches upon an area of the brain possessed of special function, *e.g.*, motor area, until it sets up a tubercular meningitis, which it usually does.

In the spinal cord, tubercular meningitis is much rarer than in the brain, and has much the same appearances, the

exudate and tubercles being most abundant over the cauda equina. It usually spreads from the brain to the cord, but it may, though it rarely does, originate in the region of the cord, *e.g.*, from a vertebral body. In this latter case its appearances are of a more chronic character.

Symptoms.—There may be a period of failing health, but the onset of the disease itself is usually sudden. There is a rigor or a convulsion, followed by vomiting, headache and some fever. The headache increases in intensity, and frequently causes the young patient to utter a short, sudden cry—the so-called hydrocephalic cry. The pulse may be rapid for a time, but soon becomes slow and irregular. There is great restlessness, and muscular twitchings are frequent. The pupils are contracted. These symptoms are due to the early exudate irritating the nerve roots. This irritation diminishes as the exudate increases in amount and causes greater compression of the brain. The vomiting and headache diminish, and the patient becomes apathetic or delirious, and gradually passes into a comatose state. The pupils become dilated, and optic neuritis and a squint of paralytic origin may appear. The head is retracted, the abdomen becomes contracted. Paralysis or spasmodic muscular contractions and general convulsions may occur. Towards the end the temperature frequently falls below normal, and the patient sinks into a typhoid state. The usual duration of the acute disease is from two to four weeks, but some cases prove fatal in a few days and others persist for months. In the latter, Kernig's sign is usually present, and Jacksonian epileptic attacks are not uncommon.

Diagnosis.—The acute onset, headache, vomiting, slow irregular pulse, the irritative, followed by paralytic, phenomena, and the eye symptoms are generally very suggestive of the disease. Choroid tubercles are seen during life far

less frequently than after death, and the *tache cérébrale* is not of much diagnostic importance, as it is of frequent occurrence in other diseases. There is generally a leucocytosis towards the end of, and often throughout, the disease. Lumbar puncture obtains a turbid fluid, which on being centrifuged shows mostly mononuclear cells (in very acute cases there may be many polymorpho-nuclears) and tubercle bacilli.

Prognosis.—The outlook is always very grave. A few cases, apparently well authenticated, are said to have recovered. There may be temporary improvement in some cases, but the vast majority march rapidly to a fatal issue.

Treatment.—The head should be shaved and an ice-bag applied to relieve pain. Lumbar puncture, originally tried for diagnostic purposes, is sometimes followed by complete, though temporary, abatement of symptoms, and is certainly a method by which intra-cranial pressure can often be reduced. There does not seem to be any corresponding benefit to compensate for the pain induced by the counter-irritation which used to be applied to the nape of the neck in almost every case. The bowels should be kept freely open.

MENINGOCOCCAL MENINGITIS.

This is caused by the *diplococcus intracellularis* of Weichselbaum. It has already been considered under "Epidemic Cerebro-Spinal Meningitis," *vide* page 77.

PNEUMOCOCCAL MENINGITIS.

The *diplococcus lanceolatus* of Frænkel or pneumococcus is the cause of this form of meningitis. It is exceptionally seen as a primary condition, there being no pneumonia or any other inflammatory lesion in the rest of

the body. Both in the character and distribution of the exudate it closely resembles the meningococcal form, but shows less preference for the base of the brain. The exudate may be serous, sero-fibrinous, or purulent, the last being the commonest. In this respect it agrees with most forms of meningitis, the tubercular being the most prominent exception, for in it a purulent exudate is rare. It is for a time a purulent lymph rather than liquid pus. It is seen in bands of a yellowish (pneumococcal) or greenish-yellow (meningococcal) colour, irregularly distributed along the sides of the larger pial blood-vessels, while the rest of the blood-vessels and general pial surface are hyperæmic and show little exudate. Later, liquid pus forms, which may penetrate to the subdural space and be widely spread over the surface of the convolutions.

Symptoms.—Pneumococcal meningitis is said to be sporadic in its occurrence, but it may occasionally appear in an epidemic way. Its symptoms so closely resemble those of the epidemic cerebro-spinal meningitis, caused by the meningococcus of Weichselbaum, or its sporadic variety, the posterior basal meningitis, caused by the modified meningococcus of Still, that a separate description of them is unnecessary.

ACUTE SECONDARY LEPTOMENINGITIS.

Etiology.—This is of far more frequent occurrence than the primary disease. It occurs in the course of rheumatic fever, tuberculosis, typhoid fever (rare), influenza, pneumonia, erysipelas, ulcerative endocarditis, anthrax, and other infectious diseases, when it is caused either by the specific germ of the primary disease or by one of the germs of common inflammation, such as the staphylococcus, the bacillus coli, &c. Mixed infections are not uncommon

during the course of a specific disease, wherein the germ of that disease acts along with one or other of the germs of ordinary inflammation. Acute secondary meningitis may follow trauma, middle ear disease, nasal disease, &c., when it is often fulminating, or as a terminal infection in chronic nephritis, heart disease, arterio-sclerosis, &c. One of the pyogenic cocci, the pneumococcus, or other similar germ is found in these cases, and mixed infections are frequent.

Morbid Anatomy.—Most forms proceed rapidly to supuration, particularly those following trauma, middle ear disease, abscess of the brain, erysipelas, &c. Some forms merely produce some congestion and œdema, *e.g.*, influenza. Œdema is the most striking feature of the form which is sometimes seen in alcoholics, so that the name “wet brain” has been given to it. In all forms of leptomeningitis there is some involvement of the grey matter of the brain in the form of exudate and cellular invasion around the blood-vessels which dip in from the pia. Many of the nerve-cells show chromatolysis. In other words, a degree of encephalitis is present in all cases of leptomeningitis, and this is most marked in epidemic cerebro-spinal and in tubercular meningitis.

Symptoms.—They are similar to those already described under epidemic cerebro-spinal meningitis, and also resemble those of tubercular meningitis, but in many of them the irritative stage is less marked and is more quickly followed by the paralytic stage. Muscular twitchings, spasms, and retraction of the neck occur when there is merely meningeal congestion without inflammation; but when headache, vomiting and delirium supervene, meningitis is to be suspected. Convulsions are not nearly so common in any of these forms of meningitis as they are in the tubercular form. Optic neuritis is most frequently seen

when the base of the brain is the chief seat of the meningitis.

Diagnosis.—The primary disease is of course the chief factor in determining the cause of the meningitis, but lumbar puncture should be made whenever possible, as it may show the bacteria actually present in the meninges.

Treatment of the above conditions.—The treatment should be on the same lines as that suggested for Tubercular Meningitis, but in certain cases operative interference is of value, and may sometimes save life. It is obviously where the initial lesion is in the middle ear that prompt surgical aid is most likely to prove beneficial.

CHRONIC LEPTOMENINGITIS.

Whitish-yellow thickened patches, irregularly distributed over the meninges, but found principally near the larger vessels of the anterior part of the brain, have been sometimes seen. They are adherent to the dura and to the cranial bones on the one side, and to the brain on the other, so that the cortex is lacerated on an attempt to strip the pia from it. The etiology of this rare condition is obscure and complicated. A chronic leptomeningitis is sometimes met with in tubercle and syphilis, and in the sporadic form of cerebro-spinal meningitis above mentioned.

Symptoms.—They are very variable, but are generally localised like those of tumor.

TUMORS OF THE PIA ARACHNOID.

Simple tumors, *e.g.*, fibroma, myxoma, osteoma, are occasionally seen. Malignant tumors, both primary and

secondary sarcomas and cancers, occur. Certain forms of primary sarcoma are not infrequent, *e.g.*, the clinically unimportant psammomas and cholesteatomas and the larger, more important forms, such as the endotheliomas and vascular sarcomas. Cancer usually shows an epitheliomatous structure. Cysts, both exudative and parasitic, occasionally occur. Syphilitic gummata and tubercular tumors usually begin in the pia mater and invade the brain.

. Symptoms.—The symptoms of pial tumors are similar to those of the brain. In their early stages irritative phenomena, *e.g.*, Jacksonian epileptic attacks, are not infrequent.

DISEASES OF THE SUBSTANCE OF THE BRAIN AND CORD.

DEGENERATIONS OF THE BRAIN AND CORD.

Cloudy swelling or granular degeneration and fatty degeneration are of very frequent occurrence in the brain, both during degenerative and inflammatory processes. So constantly and characteristically do they occur in inflammation, and so difficult is it to determine in many cases whether their causation is of a degenerative or an inflammatory nature, that it is more convenient from practical considerations to discuss them under one heading. They are therefore all included under the inflammations of the substance of the brain and cord (*vide* page 890).

Hyaline and waxy degenerations are seen chiefly in connection with the blood-vessels under circumstances similar to those in which they are met with elsewhere in the body, and in many chronic inflammations. They do not require separate consideration.

HYDROCEPHALUS.

DEFINITION.—A great accumulation of fluid within the ventricles of the brain. The term is by some extended to include also an excessive collection of fluid in the subarachnoid space, called an *external hydrocephalus* or a *hydrocephalus ex vacuo*.

External hydrocephalus is of comparatively common occurrence in varying degree in all cases of atrophy of the brain substance, *e.g.*, in old age and wasting diseases, where it merely occupies space formerly filled by the brain substance, *i.e.*, is a secondary accumulation within the subarachnoid space. But some cases occur in intra-uterine life, and in these it may be primary and the brain atrophy secondary. Some cases of microcephalus or hypoplasia of the brain may be of this nature.

Internal hydrocephalus occurs in three forms:—(1) Quincke's form, called also serous meningitis or idiopathic internal hydrocephalus or angio-neurotic hydrocephalus, (2) congenital or infantile chronic hydrocephalus, (3) acquired or secondary chronic hydrocephalus. Acquired acute hydrocephalus is a synonym for acute tubercular meningitis.

QUINCKE'S FORM.

Etiology.—Its cause is unknown, but it attacks adults chiefly, though children also suffer.

Morbid Anatomy.—The chief change is the great accumulation of cerebro-spinal fluid within the ventricles. It remains clear in appearance and its composition does not differ from that of the normal cerebro-spinal fluid. This can be demonstrated during life by lumbar puncture. This fact is important, as many of the symptoms simulate a meningitis. The lining wall of the ventricles is to all appearance normal in acute cases, though it is somewhat thickened in long-standing cases. There are no

other changes, and the pathological process seems rather to be of the nature of an œdema than of an inflammation.

Symptoms.—In the acute cases there may be fever, but it is not common. There is headache, signs of intra-cranial pressure, choked disc, slow pulse, retraction of the neck, &c. In the chronic cases the symptoms are those of a tumor. Recovery is reported in some of them, and variation in the intensity of the symptoms is a common feature.

Treatment.—It is possible by repeated lumbar puncture to drain away a limited amount of the cerebro spinal fluid and so to relieve the headache and other inducers of intra-cranial pressure. The use of mercury has been recommended, and a prolonged trial in small doses should be given.

CONGENITAL FORM

Etiology.—The cause is unknown. The disease may appear in several members of the same family. It begins in intra-uterine life, sometimes quite early, and may run its whole course before birth, or develop considerably after birth. The head may be so large as to interfere with delivery at the time of birth.

Morbid Anatomy.—The principal feature in the disease is the great distension of the lateral ventricles. It is nearly always symmetrical. The appearances and composition of the fluid seem normal, and the lining wall of the ventricles is either unaltered or a little roughened. The choroid plexus may be enlarged, pale or vascular, but is often normal. The third ventricle is also enlarged, and so is the aqueduct of Sylvius, though to a less extent. The fourth ventricle is rarely enlarged. In consequence

of this great accumulation of fluid within the ventricles (it may amount to several litres) the cerebral substance is greatly thinned, and may in the middle regions of the brain be reduced to a mere shell, measuring only a few millimetres in thickness, showing no trace of sulci or convolutions. The basal ganglia are similarly flattened. The cranial bones are widely separated, and Wormian bones develop in the sutures. These changes give a striking appearance to the child. Its head is large and globular, the forehead bulging, the face small, and the eyeballs often projecting, an exophthalmos due to the depression of the orbital plates of the frontal bones.

Symptoms.—Marked hydrocephalus is usually fatal within the first four or five years of life, but the progress of the disease may be arrested and the patient may reach adult life. The mental condition is variable, but though the child may be bright, perfect development of all the faculties is improbable. Some grade of imbecility is common. The power of walking is acquired late, and permanent muscular feebleness or spasticity is likely to appear in marked cases. Convulsions, choked disc, and nystagmus are not infrequent.

Treatment.—Tapping the lateral ventricles through the anterior fontanelle has been often recommended, and if combined with carefully applied pressure by means of an elastic bandage, success may be attained. Lumbar puncture is also a simple means of withdrawing cerebrospinal fluid. The difficulty has always been to prevent re-accumulation, and the efforts to establish continuous drainage of fluid from the ventricles into the subdural space or elsewhere are not as yet wholly satisfactory.

THE ACQUIRED CHRONIC FORM.

Causes.—Pressure on the veins of Galen by a tumor is the commonest cause. Obliteration of the iter by a tumor or by parasites is a much less frequent cause, as is also a closure of the foramen of Magendie brought about by meningitis. Inflammation of the ependyma may cause a single or double hydrocephalus of the lateral ventricle, according as it blocks one of or both the foramina of Monro.

Morbid Anatomy.—The affected ventricles are distended. Their walls may be smooth or thickened. The fluid is clear. A tumor is frequently present in the third ventricle or at the base of the brain. The brain substance may be greatly atrophied. The cranial sutures may or may not be separated. In the former case the head will gradually enlarge, in the latter it cannot do so.

Symptoms.—The evidences of pressure are more prominent than in the infantile variety. There is generally headache and vomiting, while convulsions, paralysis and gradually progressive optic neuritis are frequent. In short, the symptoms are much the same as in cerebral tumor.

Treatment.—Here the anterior fontanelle is not as a rule available and we must either trephine the skull or else try repeated lumbar puncture. Morphia is often necessary, but the symptoms are most satisfactorily relieved by withdrawing cerebro-spinal fluid.

HYDROMYELIA AND SYRINGOMYELIA.

DEFINITION.—Hydromyelia is a dilatation of the central canal of the spinal cord. Syringomyelia is a cavity formation within a gliomatous new growth in the neighbourhood of the central canal of the cord.

HYDROMYELIA.

Etiology.—The cause of the dilatation of the central canal is an increase of the cerebro-spinal fluid, but the explanation of this increase is not always clear. Some cases are congenital. In the acquired cases degenerative or inflammatory changes within or just around the ependyma may produce the increase of fluid, and it may also be due to abnormalities in the blood or lymph circulations.

Morbid Anatomy.—The dilatation of the central canal may be slight or great; its shape, which is usually rounded may be triangular, slit-like, or irregular. The canal is always lined by the ependymal cells and filled with cerebro-spinal fluid, usually unaltered, rarely hæmorrhagic or purulent.

Symptoms.—It gives rise to no symptoms so far as we know, and is therefore of no clinical significance.

SYRINGOMYELIA.

Etiology.—The cause is unknown. It is much more common in men, and most cases begin before the thirtieth year. The primary form consists in the formation of a gliomatous tissue within which solution and cavity formation occur. This gliosis may be dependent upon the persistence of embryonic tissue. The secondary variety is formed entirely by the union of myelitic or hæmorrhagic cavities or cysts around which a condensation and forma-

tion of gliomatous tissue occur as a secondary healing process.

Morbid Anatomy.—The cavity is usually irregular, and is not lined by ependymal epithelium. It is rarely situated centrally, usually to one side of or behind the central canal, which may still be visible or obliterated. It often extends into one of the posterior cornua. It may be very small or so large as to distend the whole cord, pressing upon and causing degeneration and atrophy of its nervous substance. Degenerations in the grey and white matter (both ascending and descending) are common sequels. It may contain clear fluid or a brownish gelatinous mass. It is surrounded by glia tissue. It probably begins in the cervical region, and may continue along the whole length of the cord, but is usually more limited.

Symptoms.—The development of the symptoms is insidious, but in many cases it follows uniform and well-known lines. In these classical cases there are typical sensory and motor symptoms. The sensory symptoms consist in irregular pains, chiefly in the cervical region, in a loss of the sense of pain and temperature (which may cause the patients to injure themselves) owing to the destruction of their paths of conduction in the grey matter, posterior to and around the central canal, whilst the muscular and tactile sense remains normal, the paths by which these impressions are conducted, viz., the postero-lateral columns, not being implicated. The motor symptoms consist in an atrophy of certain muscles in the arms, and sometimes also in the legs. It often begins in the small muscles of the hands and extends upwards like progressive muscular atrophy. The muscles of the legs become spastic, the tendon reflex is increased, and the Babinski sign is positive. The picture thus comes to resemble that of amyotrophic lateral sclerosis. Scoliosis may be present owing to weak-

ness of the muscles of the back. Considerable variations occur, the sensory phenomena may not appear for years after motor signs, or *vice versa*. Marked trophic changes may be present in the bones (brittleness) and joints (destruction) of the affected limb or limbs, or they may occur in the skin, nails, and tissues of the hands. The skin becomes glossy, the nails thickened and brittle. Deep-seated ulcers and painless destructive whitlows of the fingers, along with neuralgic pains and cutaneous anæsthesia of the hands and wasting and paralysis of the muscles of the hand and forearm, constitute a type known as Morvan's disease. It closely simulates in appearance the anæsthetic variety of leprosy, from which it is easily distinguished by the absence of pigmentation, and by the course which it runs. When the lumbar part of the cord is involved the organic reflexes may be abolished, and bedsores, cystitis, and incontinence of urine and fæces may arise. When the upper part of the cord and medulla is implicated respiratory and cardiac embarrassment may follow.

Diagnosis.—In the classical cases the loss of painful and thermic sensations, combined with the amyotrophic paralysis, make the diagnosis easy. Difficulty may arise in some of the variations. It is distinguished from leprosy by the lack of pigmentation and by the course and general characters of the disease, from cervical pachymeningitis by the absence of head retraction, and from a tumor of the cord by its slower progression.

Prognosis.—The disease may be steadily progressive, but it often becomes and remains stationary for many months or years, during which amelioration of many of the symptoms may take place.

Treatment.—The treatment cannot be curative, but much may be done for the relief of symptoms as they

occur, and especially those associated with bed-sores and with the bladder.

SPINA BIFIDA.

Etiology and Morbid Anatomy.—It is a congenital condition, resulting in defective closure of the bony canal containing the cord. The deficiency consists in a failure to lay down in cartilage the neural arches and spines of certain of the vertebral bodies. The spina bifida is formed by a hernial protrusion of the membranes of the cord through this opening. The sac is filled with cerebro-spinal fluid, and the spinal nerves are usually spread out over its inner surface. It is usually found in the lumbar region of the cord. The central canal of the cord may be distended, but it rarely communicates with the sac. The sac is usually covered with skin, which is hairy.

Symptoms.—There are generally no symptoms, though a certain amount of atrophy and paralysis of leg muscles may be present, of which talipes varus is the most frequent result.

Treatment.—The treatment is surgical.

CIRCULATORY DISTURBANCES.

ACTIVE HYPERÆMIA of the brain and of the cord is of frequent occurrence, but it causes *per se* little or no disturbance.

ANÆMIA OF THE BRAIN, on the other hand, is of considerable importance. It is produced in many ways, *e.g.*, from weakness of the heart muscle, from increased intra-thoracic

pressure interfering with the proper filling of the heart, from dilatation of the abdominal blood-vessels. Obstruction to the flow of blood through either of the carotid arteries, *e.g.*, ligature outside the skull, does not produce anæmia of the brain. Ligature even of both carotid arteries is not followed by ill effects, provided the tying be done slowly and a sufficient interval of time be allowed to elapse between the two operations. The symptoms of anæmia of the brain of acute onset are flashes of light or dark spots before the eyes, singing in the ears, giddiness, headache, faintness, unconsciousness and convulsions, or even death. When it is of chronic onset there may be mental weakness and irritability, faintness, headache, strabismus, and giddiness. There may be mental wandering or a semi-comatose state, or even convulsions.

ANÆMIA OF THE CORD is also of frequent occurrence. It is present in all severe general anæmias, and in profuse hæmorrhages, *e.g.*, from the stomach or uterus. It leads in many of the severe general anæmias, *e.g.*, pernicious anæmia, to degenerations in the white columns, particularly in the posterior columns, but it does not always do so, and it is doubtful if it can do so unless it is helped by the action of co-existent toxins. The influence of a sudden deprivation of the blood supply to the cord which occurs in profuse hæmorrhages may be studied experimentally by ligature of the abdominal aorta. Destructive changes, particularly in the cells of the lumbar part of the cord, result, which produce a paraplegia either at once or after an interval of some days. Death follows unless the collateral circulation is established in time, in which case the paraplegia disappears. An anæmia involving a local part of the cord is met with in compressions by tumors or inflammatory exudates, and in arterial obstructions by arterio-sclerosis, thrombosis and embolism, whose clinical results are referred to under these headings.

ŒDEMA OF THE BRAIN occurs in general œdema, in mitral stenosis, &c. A limited but very marked form is seen around tumors and abscesses. Its appearances are similar to those of anæmia. The surface of the brain and its membranes are pale, the blood-vessels being empty. The convolutions are atrophied, and there is an increase of cerebro-spinal fluid in the sulci and in the subarachnoid space. On section, the brain substance, both grey and white matter, is pale, with a moist glistening appearance. The symptoms are not well defined. Cases in which it occurs show anæmia, and generally some condition of intoxication as well, so that it is difficult to say what symptoms the œdema by itself may cause. They are probably similar to those of anæmia. Uræmia has been attributed in part to it, and some writers have stated that it may cause hemiplegia.

ŒDEMA OF THE CORD occurs under similar circumstances but is rarely so marked as in the brain.

RUPTURE OF VESSELS.

CEREBRAL HÆMORRHAGE.

INTRODUCTION.—Hæmorrhage on to the surface of, or into, the membranes is called *meningeal*; into the substance of the brain, *cerebral*, or *apoplexy*. This is the commonest cause of apoplexy, which means simply a sudden stroke. Other causes are embolism and thrombosis. *Meningeal* hæmorrhage is most frequently caused by fracture of the skull, and the vessel most often torn is the middle meningeal artery. The blood collects between the dura mater and the bone, and causes compression of the brain. A torn sinus is an occasional source of the hæmorrhage. A more frequent source is the rupture of an aneurism of

one of the cerebral arteries. These vessels lie in the subarachnoid space, and hence the blood permeates into that space, usually also into the subdural space, and sometimes into the region of the cord. In most cases, when the seat of the aneurism is at the base of the brain there is more or less extensive laceration of the brain substance as well. Other and less common sources of meningeal hæmorrhages are the bursting of an intracerebral hæmorrhage into the meninges, its occurrence during the course of infectious fevers due to toxic action, and in the new-born due to injury during birth. *Cerebral hæmorrhage* is most frequently caused by rupture of one of the basal group of vessels (the name given to the arteries forming the circle of Willis), the central arteries passing from it and from the first part of the cerebral arteries. It is one or other of these central arteries which is most commonly affected, particularly those given off by the middle cerebral in the anterior perforated spaces. One of the largest of these central or nutrient branches of the middle cerebral—the *lenticulo-striate*—which goes to the third part of the lenticular and to the anterior part of the internal capsule, is so often ruptured that Charcot nominated it the artery of cerebral hæmorrhage. The lenticulo-thalamic artery stands next in frequency, and together these two branches account for over 60 per cent. of all cerebral hæmorrhages. The commonest site of the hæmorrhage is therefore the cerebral basal ganglia and their adjoining nerve tracts, particularly the internal capsule. The pons is the next commonest site, after which comes the cerebellum, and those other parts of the cerebrum, except the cortex, which is very rarely the seat of spontaneous massive hæmorrhage. The medulla is also a very rare seat.

Etiology.—Disease of the vessel wall is the main factor in the causation of the hæmorrhage. It may

involve the outer, middle, or inner coats alone, or in combination; but while it varies in nature, it almost always gives rise to minute multiple aneurisms (about the size of a pin's head), rupture of one of which is the immediate source of the hæmorrhage. In a few cases no aneurisms large enough to be detected by the unaided eye are to be seen, and the diseased vessels are believed to rupture without the previous formation of visible aneurisms. The vessel walls are feebly supported by the brain tissue even when healthy, and still more feebly when foci of softening occur around the vessels. Marie believes that such foci, which he called *lacunæ*, varying in size from a pin's head to a pea, are of common occurrence in people over sixty years of age. They favour both the production of aneurism and rupture. Cerebral hæmorrhage is most frequently met with in elderly people, *e.g.*, after fifty, for that is the period in which chronic arterial disease prevails, but it may appear much earlier, particularly in those hereditarily disposed to it. Accordingly, massive cerebral hæmorrhage occasionally occurs in children. The influence of unusual muscular efforts, *e.g.*, lifting a heavy weight or running, must not be forgotten. Thus many cerebral hæmorrhages occur during straining at stool, the weakened vessels being unable to stand the suddenly increased blood-pressure. This general arterial disease is not always present. An embolus, particularly if infective, may locally weaken the arterial wall sufficiently to cause it to rupture. Hence there is a history of rheumatism or other cause of endocarditis in some cases of cerebral hæmorrhage, particularly in the young.

Morbid Anatomy.—The escaping blood ploughs up the brain tissue, and causes further hæmorrhage by tearing fresh blood-vessels in its course. It often makes its way into the lateral ventricles, and sometimes, though rarely, through the

cortex into the meninges. Coagulation occurs, and a varying sized mass of ordinary dark red blood-clot is formed, surrounded by hyperæmic brain tissue, in which there may be many punctiform hæmorrhages. Pressure is thus exerted upon every part of the brain, though it is greatest in the hemisphere which is the seat of the hæmorrhage. The cerebral convolutions are flattened, particularly in this hemisphere, the sulci are obliterated, and the cerebro-spinal fluid diminished. As already mentioned, the hæmorrhage most frequently occupies the region of the lenticular nucleus, the internal capsule, the nucleus caudatus, or the optic thalamus. It is rarely confined to either the white or grey matter. Its size and extent determines the amount of brain tissue destroyed, and large hæmorrhages may rupture into the ventricle or extend out as far as the island of Reil, and even destroy the greater part of the hemisphere. If the patient survives, the serum is absorbed and the clot disintegrated. The hæmoglobin dissolves out of the red cells and is in large part absorbed by the surrounding vessels, to be in time excreted by the urine. It stains the brain tissue around the clot of a reddish-yellow colour. Part of it is deposited here in a solid form as hæmatoidin crystals. The stroma of the red cells and the fibrin of the clot undergo fatty degeneration and gradual absorption, leading to a chronic inflammatory reaction in the surrounding brain tissue. When disintegration and absorption are complete (which takes many weeks or months to be accomplished), a cavity containing fluid, walled in by sclerosed brain tissue, frequently results—the so-called *apoplectic cyst*. When the hæmorrhage is cortical, the outer wall of the cyst is formed by the thickened meninges. This always occupies a smaller area than the original hæmorrhage. Occasionally there is no fluid, but a general formation of scar or fibrous-looking tissue throughout the whole area. In addition to these changes at the site of the hæmorrhage itself, other degenerative

lesions of great importance arise in the nerve tracts involved. For example, if the motor strands in the anterior two-thirds of the posterior limb of the internal capsule are destroyed by the hæmorrhage, their distal parts, being permanently cut off from their trophic centres in the cerebral cortex, will progressively degenerate throughout their whole course from the internal capsule through the pons, medulla and pyramidal tracts of the cord to their terminations in the cells of the anterior horns. These secondary degenerations depend entirely upon the position of the hæmorrhage. They must follow upon all destructions of nerve-cells and nerve-fibres, for there is never any regeneration thereof in the central nervous system, and the whole part of the axones severed from their central cells must inevitably perish.

Symptoms. — Certain symptoms appear immediately, from the destructive action of the hæmorrhage; others come on gradually as the result of the secondary degenerations. Hence it is convenient to divide the symptoms, like the lesions, into primary and secondary.

The PRIMARY SYMPTOMS.—Premonitory symptoms are rare. Sensations of numbness, tingling or pain in the limbs, or disturbances of vision, occasionally give warning of an impending attack. *Loss of consciousness* and *paralysis* are the chief symptoms characteristic of the attack itself. *Loss of consciousness* always occurs except in small internal and in cortical hæmorrhages. It may be instantaneous, causing the patient to fall from the sitting or standing posture, but it is often of more gradual onset, taking a few minutes to become complete. Less frequently there is a still more gradual development, several hours elapsing before it becomes complete (ingravescent apoplexy). Sometimes it escapes notice altogether, either from its occurrence during sleep or from its slight and transient character. The appearances presented by the

patient when completely unconscious are striking. The face is usually of an ashen-grey hue, occasionally it is congested. He cannot be roused, and breathes slowly and in a laboured way, blowing out both cheeks, the paralysed one more noticeably, during expiration, and often making a spluttering noise with his lips. The breathing may be of the Cheyne-Stokes character. The condition of the pupils varies, dilatation is most common, but inequality is frequent. When marked contraction is present, the hæmorrhage is generally into the pons or the ventricles. The pulse is slow and full, the temperature normal or subnormal. A high temperature occurs in certain basal hæmorrhages. The reflexes are lost whilst unconsciousness lasts. The urine and fæces are passed involuntarily. Convulsions may occur at the outset, but they are not common. The duration of the unconsciousness is very variable. It may be quite transient or last for hours or days. In favourable cases it lessens or disappears early, or not later than the third or fourth day, whereas if it persists and deepens during these days the outlook is unfavourable. The *paralysis* is usually abrupt in onset, though it sometimes takes several hours to be established. It may affect the face, arm or leg, generally all three. It is sometimes so slight in old people as to amount merely to a dragging of the leg. In more severe cases the affected limbs are seen even, during the unconscious stage, to be more flaccid and flattened than those of the other side. When raised, they fall more quickly than the latter. The head and eyes are turned away from the paralysed side (conjugate deviation). The distribution of the paralysis depends upon the seat of the hæmorrhage, *i.e.*, upon the particular part of the brain which has been destroyed. It can only be fully determined after consciousness is restored. A few instances may be given. Destruction of the cortex in the lower (face), middle (arm), or upper (leg) parts of the ascending

parietal convolution on one side of the brain causes paralysis of motion in the face, arm or leg respectively of the opposite side of the body—a *cerebral monoplegia*. Destruction of the motor tracts in the anterior two-thirds of the posterior limb of the internal capsule on one side causes paralysis of motion in the lower part of the face, of the arm, and of the leg, all three on the opposite side of the body—a *hemiplegia*. The muscles of the upper part of the face may be weakened, but they are not paralysed. The patient can close the eye on the affected side, though not so firmly as on the other, and the upper eyelid may droop slightly. The tongue, when protruded, deviates towards the paralysed side, owing to the unopposed action of the genio-hyoglossus muscle of the sound side. The conjugate deviation, usually first seen (viz., head and eyes turned away from paralysed side), may pass off soon or be replaced by another in the opposite direction. The muscles of the thorax and abdomen appear to be little if at all affected. The paralysis is generally more complete in the arm than in the leg. This difference becomes more marked during convalescence, since some of the muscles primarily paralysed recover their tone. This is explained partly by relief of pressure and partly by innervation. Tracts merely compressed (not destroyed) by the hæmorrhage regain their function after removal of the pressure. The influence of innervation is more complicated. Broadbent's explanation seems to be the best. It divides muscles into three groups:—(1) those habitually innervated from both hemispheres, viz., the muscles associated with symmetrical movements like those of the thorax and abdomen, are not paralysed; (2) those habitually innervated from one, but anatomically connected with both hemispheres, like many of the leg muscles, recover their lost power early; (3) those habitually innervated from one hemisphere, and anatomically connected only with it, like many of the

muscles trained for delicate movements, *e.g.*, of the hand, remain paralysed.

The great majority of cerebral hæmorrhages take place into the anterior two-thirds of the posterior limb of the internal capsule, causing a typical *hemiplegia*, whose characteristics have therefore been given in detail. Small hæmorrhages in this situation may involve the genu, and only slightly the adjacent parts of the anterior and posterior limbs, when the paralysis will be limited to the muscles of the tongue, mouth, and shoulder; or the anterior third of the posterior limb, when the paralysis affects the arm; or the middle third, when it affects the leg. On the other hand, if the hæmorrhage involves the posterior third, it causes loss of *sensation* on the opposite side of the body. These are all examples of *ordinary hemiplegia*; but hæmorrhages below the internal capsule usually cause a *crossed hemiplegia*, *i.e.*, loss of function in one or more cranial nerves of one side, along with loss of motion or sensation in the opposite side of the body. For instance, hæmorrhage into the crus causes paralysis of the leg, arm and lower face on the opposite side, and paralysis of the oculo-motor nerve on the same side. Hæmorrhage into the lower part of the pons, if small and unilateral, causes hemiplegia of the opposite side and paralysis of the face on the same side from involvement of the seventh nerve; if large, there may be convulsions, followed by bilateral paralysis.

This account of the early local results of hæmorrhage takes into consideration only the more frequently occurring ones. Those arising in other situations will give similar results, dependent upon the functions of the parts destroyed. There are, however, other early symptoms in all hæmorrhages which are not local but general, being caused by the constitutional reaction to the absorptive and inflammatory changes which arise in and around the hæmorrhage. They may appear as early as two to four hours, and usually

within forty-eight hours, after the onset of the hæmorrhage, and may continue for a week or more, though they occasionally last as long as two months. They are muscular rigidity, called early rigidity, in the paralysed limbs, trophic changes, such as vesicular eruptions or the formation of a bed-sore, and congestion at the bases of the lungs.

The SECONDARY SYMPTOMS.—Muscular rigidity in the affected limbs, called late rigidity, appears in a few weeks. It is due to the secondary descending degeneration in the pyramidal tracts of the cord, and is most marked in the arm. The attitude which the arm gradually assumes is characteristic. The forearm is permanently flexed upon the upper arm, the hand upon the forearm, and the fingers upon the hand. The leg is much flexed at the knee or swung outwards in a semicircle in walking to prevent the toes dragging upon the ground. The muscles do not atrophy, or only do so slightly, as a rule. The skin of the affected limbs becomes soft and glossy. There may be tremor, choreiform or athetotic movements on the affected side, or disease of the joints. The late rigidity occasionally fails to appear, particularly in children, when there will be no contracture. In these cases the leg recovers completely, but the arm only partially and remains flaccid. This is known as “hemiplegie flasque” of Bouchard. The deep reflexes are exaggerated, while the superficial reflexes are usually diminished. Thus the knee-jerk is increased, and ankle clonus may be present on the paralysed side. An exceptional superficial reflex is frequently present, known as Babinski’s sign, which consists in extension, *i.e.*, dorsal flexion, of the great toe upon stimulation of the skin of the sole of the foot. It is an early symptom, appearing generally long before the exaggeration of the deep reflexes.

Diagnosis.—The unconsciousness and the paralysis are the chief guides, but while they make the diagnosis clear

in many cases they fail in others. When they are of sudden onset, the cause may be either hæmorrhage or embolism, when of gradual onset (a few hours to two days) the cause may be either hæmorrhage or thrombosis. It is impossible to differentiate between these causes in many cases. But embolism and thrombosis are not the only causes of difficulty in diagnosis. Certain cases of uræmia, diabetes, alcohol and opium poisoning also present difficulty, and it is of great practical importance that the differentiation should be made in such cases. The coma of apoplexy is profound and there is complete muscular relaxation. Early rigidity or conjugate deviation may come on and help to a diagnosis of hæmorrhage, but when it does not, and when there is complete relaxation, as in hæmorrhage into the ventricles, it is difficult to say whether a hemiplegia is present or not. The odour of the breath, e.g., alcohol or acetone, may help, but it is important to remember that, either through habitual use of alcohol or its administration just before the onset of an attack, its odour may be present in the breath of a patient suffering from an apoplectic stroke. The previous history of the case and the method of onset both help. Alcoholic coma usually sets in more gradually and is less intense than that of apoplexy. Uræmic and diabetic coma are usually much more gradual in development. In opium poisoning the pupils are often strongly contracted, whereas they are mostly dilated or unequal in cerebral hæmorrhage, except into the pons. Pin-point pupils suggest both opium poisoning and pontine hæmorrhage. The temperature is low in the former and raised in the latter. In all cases of coma the head should be examined for injury, and the urine for albumin, sugar and casts; the odour of the breath, the state of the pupils and the muscles, and the history of the case must be ascertained before a diagnosis is made, and even then it should be made reservedly. Undue weight must not be placed upon any single fact:

thus the odour of the breath may mislead, since drunkards often suffer from apoplexy, and signs of renal disease may do the same, since renal disease may lead to uræmia or cerebral hæmorrhage.

Prognosis.—Large internal hæmorrhages, particularly when they rupture into the ventricles, prove rapidly fatal. The following are unfavourable signs:—persistence without any lessening of the coma for over twenty-four hours, its persistence and deepening during the second and third day, an early rapid rise of temperature, considerable fever, or the formation of bed-sores during the period of reaction. In favourable cases some amount of permanent paralysis and contraction are to be expected, except in cortical hæmorrhages, which usually, unless when they are extensive, recover completely; but infantile meningeal hæmorrhages may produce idiocy or spastic diplegia.

Treatment.—As soon as possible the patient should be placed in bed, and the clothes surrounding the neck loosened. An attempt should be made to determine whether the hæmorrhage is *meningeal* or *intra-cerebral*. If it is *meningeal*, trephining is of great value, because not merely may the hæmorrhage be arrested, but clots may also be removed.

The majority of cases are *intra-cerebral*, and probably one of the most important points to decide is whether the blood pressure is unusually high, because, if so, free purgation, diuresis, and even blood-letting may be necessary. The best purgative to give to an unconscious patient is, unquestionably, croton oil, in a dose of one to two minims, mixed with one or two drops of glycerine or olive oil, and placed on the back of the tongue. Blood-letting is of special value where there is evidence that the hæmorrhage is progressing. As a rule, the median basilic vein is selected, and ten to twenty ounces of blood removed. According

to Cushing, the blood pressure is greatly raised by the hæmorrhage, and he suggests that it may have a salutary influence in maintaining the patient's vital processes. This evidence, derived in part from experimental work on animals, militates against venesection, although where the hæmorrhage is progressing it does not justify the physician in entirely neglecting the practice of phlebotomy. The proper nursing of the patient demands much care. There is a great tendency to the development of bed-sores, and particularly if the skin is allowed to remain soiled with urine or fæces. The most careful cleansing operations should follow the evacuation of bowels or bladder, and care in connection with this matter is amply repaid. The bladder should be watched in case it becomes over-distended.

Wrap up the paralysed limbs in wadding, and lightly bandage until, at all events, some days have elapsed. Once the stage of reaction has passed off, massage, together with movements of the limbs, should be regularly carried out, and faradism has certainly a beneficial effect in keeping up the nutrition of the paralysed muscles. The diet should be simple, mostly milk puddings, and only to a small extent should soups and butcher meat be administered to the patient.

Contracture is very troublesome, and may come on notwithstanding the greatest care. It should be obviated, as far as possible, by massage and passive movements. These must not be too long continued on any one day, although they should be kept up for months.

For the stage of reaction, absolute rest must be enjoined, purgatives and diuretics administered to the patient, and an ice-bag applied to the head if the temperature is high and excitement considerable.

The later treatment of a case of cerebral hæmorrhage varies considerably. Should there be any suspicion of syphilis, administer potassium iodide, and possibly mercury,

and it is conceivable that in any case the iodide may help the absorption of blood clot. In old and feeble people, remember the great importance of preventing hypostatic congestion of the bases of the lungs. There is no question that raising the patient from time to time, and altering his position in bed, tends to obviate its development. Do not be discouraged if the patient remains unconscious for even five or six weeks, because in the most hopeless looking cases remarkable improvement may set in, and at least life may be prolonged for years, although with considerable permanent paralysis.

Later on an attempt may be made to prevent deformity where more or less complete recovery has not occurred. To this end, section of tendons and transplantation have sometimes been tried with good results.

SPINAL HÆMORRHAGE.

The hæmorrhage may be into the spinal membranes, called *Hæmatorrhachis* or meningeal apoplexy, or into the substance of the cord, called *Hæmatomyelia*.

HÆMATORRHACHIS.

Hæmatorrhachis may be extra- or intra-meningeal, *i.e.*, either outside the dura and between it and the vertebral column, or inside the dura in the subdural or subarachnoid space.

Etiology.—The causes of extra-dural hæmorrhage are either trauma or the rupture of an aneurism of the aorta which has eroded the vertebral bodies. The causes of hæmorrhage internal to the dura are much more various. Injury to the spinal column is one of them, though less frequently than it is of the extra-dural variety. Injury to the skull is also a cause. Fracture of the base of

the skull often results in blood flowing downwards within the subdural or even subarachnoid space, particularly on the posterior aspect of the cord. Its presence here in such cases during life may be demonstrated by lumbar puncture, and this procedure may aid diagnosis. Rupture of an aneurism of one of the cerebral vessels at the base of the brain is a cause of large hæmorrhage within the membranes of the cord. Hæmorrhage into the substance of the brain, when it ruptures into the lateral ventricles, flows through one of the foramina of Monro, the third ventricle, the iter, and the fourth ventricle, and thus downwards for a considerable distance of the cord within the subarachnoid space. Small focal hæmorrhages occur in the acute infectious fevers and in death from convulsive diseases such as epilepsy, tetanus and strychnine poisoning and difficult parturition. Hæmorrhage occurs also in the hæmorrhagic form of pacchymeningitis interna.

Symptoms.—The onset is usually abrupt. When the hæmorrhage is great it may soon cause death by pressure. In severe cases not proving quickly fatal there is paralysis of the legs, of the trunk, or of the arms, according to the site of the hæmorrhage. Sharp pain and other evidences of irritation of the spinal nerve roots and muscular spasms may precede the paralysis, particularly when the latter develops slowly and remains incomplete. The organic reflexes are abolished when the lumbar region is involved. Trophic disturbances, such as herpes, may be present.

Diagnosis.—The history of the case, the mode of onset, and the character of the symptoms, aided by lumbar puncture, generally enable a correct diagnosis to be made.

Prognosis.—Recovery takes place in some cases, particularly in those in which the amount of hæmorrhage is

small, as in the milder traumatic and infectious cases, but a fatal issue is to be feared in most cases. Generally the higher up the cord the site of the hæmorrhage is, the more serious it is.

Treatment.—In traumatic cases operative interference is certainly indicated. In cases of aneurism and in hæmorrhages into the membranes due to other causes it is unfortunately rarely possible to do more than treat symptoms.

II. HÆMATOMYELIA.

Etiology.—Traumatism is the most common cause, hence the disease is more common in males. Bullet wounds, fractures and dislocations of the vertebral column, or even acute and forcible bending of the neck without fracture, are the most frequent among such injuries. Hence the cervical region of the cord suffers oftenest. A hæmorrhage within the substance of the cord, as in the brain, may take its origin within the substance of a glioma. Small hæmorrhages occur also in tetanus, strychnine poisoning, and other convulsive diseases, in diffuse, focal and pressure forms of myelitis, in degenerated areas, in acute infections, and even in general passive congestions.

Morbid Anatomy.—The site, size and shape of the hæmorrhage vary greatly. The grey matter suffers more frequently than the white, and small hæmorrhages may be limited to it. Usually both are involved and the blood may spread transversely and rupture into the central canal or through the surface of the cord into the subarachnoid space, or it may spread longitudinally up the white strands for considerable distances. It usually affects only one side of the cord. The immediate and remote changes in the clot itself, in the cord substance around the clot, and in the ascending and descending tracts of the cord are similar to those of hæmorrhages into the brain.

Symptoms.—They vary greatly, according to the site and size of the hæmorrhage. When limited to one side of the cord, which is common, they tend to produce a type of the Brown-Séquard paralysis, *vide* page 843. The onset is sudden. Death is immediate when the hæmorrhage is bilateral and high enough to involve the centres for the diaphragm. In other severe cases death may be delayed for a time, but usually supervenes upon an extension of the myelitis. Many cases survive with some motor and sensory arm or leg paralysis and some degree of spasticity in the leg.

Diagnosis.—The history of the case, the rapid onset, and the character of the symptoms often make the diagnosis easy, but there may be much difficulty in the small focal hæmorrhages of inflammation where fortunately its diagnosis, apart from the myelitis, is not of practical importance.

Treatment.—Rest and attention to bladder and skin are the chief indications for treatment. Severe cases are so rapidly fatal that no treatment is possible.

CAISSON DISEASE—DIVER'S PARALYSIS.

This disease, which occurs in men subjected to high atmospheric pressures, such as divers and workers in caissons, is best considered here, as it is most closely allied to hæmorrhage into the substance of the cord.

Etiology.—The disease is caused by subjection to an atmospheric pressure of more than three atmospheres for too long a time and a too rapid release therefrom. The compression is believed to cause a greatly increased absorption of gases by the blood and tissues, according to

Dalton's law until complete saturation occurs. Vernon asserts that the fatty tissues absorb at least five times as much as does the blood and other tissues. Hence the spinal cord, which is rich in fat-like substances, absorbs a disproportionately large quantity. On too rapid decompression it is thought that the dissolved gases, chiefly nitrogen, not being given time to escape naturally, collect in bubbles in the blood, constituting gas emboli, and in the tissues, particularly in the spinal cord and liver, tearing and lacerating them. Inexperienced workmen are said to suffer most, stout more than spare men, and also those under par as the result of alcohol or chronic heart or kidney disease.

Morbid Anatomy.—Hæmorrhages and myelitis have been found in fatal and in experimental cases, also rents and fissures in the cord, liver, &c., the surrounding cells being compressed, but these changes are not constant.

Symptoms.—Agonising pains come on first in the joints, then in the muscles of the limbs, back, and abdomen. Vomiting is common. Headache, giddiness, faintness, and paralysis may follow, but are less frequent. These symptoms generally supervene immediately on leaving the caisson, or they may be delayed for several hours. The patient may indeed become rapidly comatose and die in a few hours, but this is rare. Most cases, even where there is paralysis, recover in a day or two, although they may take several weeks or months to do so. The work of Hill and Macleod, of Haldane and Boycott has considerably increased our knowledge of this disease, and it is probable that serious symptoms may be largely if not completely avoided in the future by recompression at once when symptoms appear, and by gradual decompression.

Treatment.—The recompression just referred to is the best method of immediate treatment. Under an air

pressure somewhat less than that in which the patient was working the pains rapidly cease and improvement sets in. The pressure can then be gradually reduced as cure is effected. Opium or other sedative is indicated for the pains where necessary, and massage and electrical treatment for paralysed muscles.

No workman out of health should be allowed to go into the caisson, and new hands should work for very short periods to begin with.

EMBOLISM AND THROMBOSIS.

A.—IN THE BRAIN.

Etiology.—*Embolism.*—The emboli come most frequently from vegetations upon the cardiac valves; the mitral oftener than the aortic. At other times they come from the left auricular appendix, the pulmonary veins, aneurisms or atheromatous plaques in the aorta. *Thrombosis* occurs in syphilitic and other forms of endarteritis, in aneurisms, and often in atheroma.

Morbid Anatomy.—Emboli are said to pass more commonly into the left middle cerebral artery, which is consequently blocked either at its origin or beyond the point of origin of its central branches. The embolus may pass on into one of its cortical branches, viz., that supplying the third frontal convolution, that going to the ascending frontal, to the ascending parietal, to the supra-marginal and angular gyri, or to the upper temporal convolutions. Thrombosis occurs oftenest in the middle cerebral and the basilar arteries. The morbid changes are the same, whether induced by embolism or thrombosis, and result in *softening* in the areas supplied by the blocked arteries. The nerve-cells and fibres rapidly undergo fatty degeneration and disappear, the neuroglia cells become

swollen and fatty, and the whole patch becomes infiltrated with fluid, the result of a colliquative necrosis. Its colour is a dead white when the white matter is implicated, as in blockings of the central branches of the middle cerebral; but red at first, becoming yellow later when the grey matter of the cortex is affected. The anastomoses of the cortical arteries are sufficiently free to permit of a certain amount of the engorgement seen in red infarcts. Red, yellow or - white infarcts or necrosed areas are thus produced. They may show very little further change for a considerable time, but usually absorption occurs within them and inflammatory reaction around them. These changes result in scar formation when the patches are small, and in false cysts when they are large. When the embolus contains pyogenic germs the necrotic focus may become an abscess.

Symptoms.—The symptoms depend upon the part of the brain affected. When the lesion implicates the internal capsule and the other parts specified under hæmorrhage, the symptoms are practically the same as therein described. Embolism may be distinguished by its onset being more sudden than in hæmorrhage, and by the existence of heart disease. Thrombosis is generally of more gradual onset, giving time for certain premonitory symptoms, such as giddiness, loss of memory, embarrassment of speech, and disturbances of sensation, *e.g.*, headache (one-sided or general, and worst at night) or tingling or numbness in the fingers. Consciousness is generally much less impaired, and may be even unaffected in thrombotic cases due to syphilis. Abrupt and deep loss of consciousness is rare in embolism or thrombosis, whereas it is common in hæmorrhage. Right-sided hemiplegia is generally associated with aphasia. In some cases there are no symptoms when indifferent or silent parts of the brain are affected.

B.—IN THE CORD.

Endarteritis is common, both in the larger and smaller branches of the spinal arteries, particularly in syphilis, but it rarely if ever causes aneurism, either miliary or larger. Emboli, with the exception of the minute infective ones, rarely involve the vessels of the cord. Thrombosis, on the other hand, is common in connection with endarteritis and in many of the acute and chronic changes in the cord.

Treatment.—The treatment for *embolism* closely resembles that recommended for hæmorrhage, only there is not the same necessity for reducing the blood pressure. In cases where aphasia develops, much may be accomplished by the careful and painstaking re-education of the patient, training the centres on the opposite side of the brain to take up the functions abrogated.

For *thrombosis*, cardiac tonics may be required, and it is well to remember that in many cases the treatment for syphilis is necessary, and that a protracted administration of potassium iodide sometimes yields fairly satisfactory results.

HEMIPLEGIA IN CHILDREN.

Etiology.—The cause is obscure. Osler gives an analysis of 135 cases. The disease appears during the first or second year of life in the great majority of cases, and is rare after the fifth year. There is a history of a difficult labour in some cases, of one of the infectious fevers in others. Hæmorrhage, embolism or thrombosis has occurred in others, whose age was usually over six.

Morbid Anatomy.—The most important changes found were—(1) atrophy and sclerosis involving varying sized areas, from groups of convolutions (particularly

those supplied by the middle cerebral artery) to an entire hemisphere, (2) cavities of varying size and depth, roofed by the arachnoid, bounded internally by the pia mater, and containing cerebro-spinal fluid. This condition is known as *porencephaly*. It is also seen in adults after embolism and hæmorrhage. Areas of softening were present in the hæmorrhagic, embolic and thrombotic cases, which constituted about twelve per cent.

Symptoms.—In the great majority of cases the onset was sudden, and it is difficult to understand why, seeing that the nature of the lesion is chronic in about seven-eighths of the cases. It is to be remembered, however, that, while the lesions found *post mortem* have all the appearances of chronicity, they may have started as inflammatory changes of acute and sudden onset, *e.g.*, an acute poli-encephalitis. More than half the cases began suddenly with severe convulsions and loss of consciousness, the latter lasting from hours to days. The convulsions may be general, but are often unilateral, involving the side which is subsequently found to be paralysed. The hemiplegia generally develops rapidly, but sometimes slowly, and on the right side it may be associated with aphasia in children who have learned to speak. Sensation is rarely affected. Fever and vomiting are frequent. The subsequent history of favourable cases shows a general improvement in the muscular power, and occasionally complete recovery thereof, the paralysis entirely disappearing. Improvement is always greater in the leg than in the arm. Late rigidity comes on in nearly all cases, and there may be jerking choreiform movements (*post-hemiplegic chorea*) or athetosis, *i.e.*, involuntary rhythmical movements of the fingers and toes. Instead of being at rest, they are constantly being flexed and extended, abducted and adducted, supinated and pronated, often in regular sequence. Impairment of mental or of

bodily development is common, and epileptic seizures frequent.

Diagnosis.—The age of the patient, the sudden onset, and the development of hemiplegia make the diagnosis clear in most cases. A cerebral tumor may sometimes give cause for doubt, but the slow development of the hemiplegia which it exhibits is generally sufficient to make the distinction clear.

Treatment.—In a certain proportion of cases trephining and the removal of blood clot from the meninges may cure the case. Unfortunately, in too many instances, nothing can be done save attention to the general health, and the use of mechanical supports to the paralysed muscles. It is impossible in cases of athetosis to do anything to arrest the jerking movements.

ANEURISMS OF THE CEREBRAL ARTERIES.

INTRODUCTION.—This refers to the single aneurism of the cerebral arteries, which run mostly within the subarachnoid space, not the multiple miliary aneurisms within the brain substance.

Etiology.—Endarteritis and embolus are the chief causes which weaken the vessel walls. Sex has the same influence here as in the production of aneurisms elsewhere in the body, a greater number occurring among men than women. Age is not the important factor it is in miliary aneurisms, since they occur as often before forty as after it.

Morbid Anatomy.—The aneurism varies from the size of a pea to that of a walnut. It occurs most frequently

on one of the middle cerebral arteries, the basilar coming next in order. The hæmorrhage may be chiefly meningeal or intra-cerebral, and sometimes it is both.

Symptoms.—Irritative or paralytic symptoms may arise prior to the rupture of the aneurism from pressure upon important structures, *e.g.*, upon the optic or other cranial nerve. The course is, however, usually latent up to the time of rupture, when the symptoms of apoplexy come on. Such cases are almost always fatal.

Treatment.—Curative treatment is impossible, and indeed it is only in an exceptional case that an antemortem diagnosis is made of the existence of the aneurism. In these cases, undoubtedly, treatment by potassium iodide should be persevered with.

THROMBOSIS OF THE CEREBRAL SINUSES AND VEINS.

Etiology.—It is generally regarded as being *primary* when it arises in cases of disease elsewhere than in the head, and as *secondary* when it is the result of a spread of inflammation from some contiguous organ, such as the ear. Primary cases occur only in the exhausted or marantic state following upon wasting diseases, *e.g.*, in weakly infants, in cases of chlorosis and anæmia, and in the later stages of cancer and phthisis. The superior longitudinal sinus is most frequently affected. Secondary cases are more common. Inflammations of the scalp (*e.g.*, erysipelas), of the bone, of the meninges, and of the brain substance are the causes. Of these, disease of the ear is the most frequent. The lateral sinus is most often affected.

Morbid Anatomy. — The walls of the superior longitudinal sinus are not much altered, and the contained thrombus is pale, firm, stratified and adherent. The walls of the lateral sinus are softened and swollen, and the contained thrombus is red, soft and crumbling, and often purulent. The process often spreads to the cerebral and jugular veins.

Symptoms. — In the primary cases the symptoms vary. There may be none, or there may be headache, mental dulness, delirium, convulsions, vomiting, paralysis (hemiplegia sometimes), as well as swelling and pain around the veins of the neck or the leg. In the secondary cases the symptoms are those of pyæmia.

Treatment. — Where the lateral sinus is thrombosed secondary to middle ear disease, first ligature the jugular vein on the affected side, and then the sinus should be opened into and cleared out. In the case of other sinuses, although thrombosis may be suspected, it is usually not necessary or possible to adopt any curative measures.

INFLAMMATION OF THE SUBSTANCE OF THE BRAIN (ENCEPHALITIS) AND SPINAL CORD (MYELITIS).

INTRODUCTION.

When the grey matter is affected the terms poli-encephalitis and poliomyelitis are used respectively for the brain and cord. The chief phenomena of inflammation exhibited by nervous tissue are the same as those shown by other tissues, viz., dilatation of blood-vessels, exudation of fluid, emigration of leucocytes, escape of red cells, and degeneration of the fixed tissues, but the course they run often

makes the recognition of the process difficult. The nerve-cells and nerve-fibres are so delicate and so prone to degeneration and death that under the action of an irritant they may show changes which from their extent and the time of their occurrence are not in keeping with inflammatory reactions in ordinary tissues. Degeneration and disappearance of nerve-cells and nerve-fibres are in all cases prominent. Dilatation of blood-vessels is frequent, and an excess of lymph exudation is probably general, but emigration of leucocytes is mostly of a limited kind and the escaped cells remain within the perivascular lymphatic sheaths, wandering but little into the surrounding nervous substance. Escape of red cells, both by diapedesis and by rupture, is an unusually common occurrence. Multiple hæmorrhages occur so frequently in the affected areas that it is exceptional to meet with an encephalitis or a myelitis free from them. In some cases the degeneration and disappearance of the nerve elements may be seen almost alone, and it is difficult to separate such cases from those of a purely degenerative process. Fortunately it is unnecessary to do so, for it is sufficient for the purposes of practical medicine to consider all such cases under one heading, that of inflammation. The onset and course of the disease may be acute or chronic.

ACUTE ENCEPHALITIS.

Causes.—They may be grouped under three heads:—
(1) Injury, (2) intoxication, (3) infection. Fracture of the skull is the commonest of the injuries, but punctured wounds and concussion require mention. The punctured wound may be made by a bullet or a sharp instrument. Intoxication is exemplified by food or gas poisoning, by lead poisoning, and perhaps also by alcohol. The infections constitute the great majority of the causes of

encephalitis, the micro-organism reaching the affected area by the circulation. The pyogenic processes, typhoid fever, influenza and epidemic cerebro-spinal meningitis afford examples. This classification of causes is a practical, rather than a strictly accurate and scientific one, seeing that many of the traumatic and toxic cases subsequently become also infective from the entrance into the damaged brain tissue of germs carried by the circulation. Some cases, particularly the milder traumatic ones, *e.g.*, in concussion, do not become infective. They quickly subside, and leave behind small foci of softening or sclerosis.

Morbid Anatomy.—The encephalitis is more frequently focal than diffuse, more often in the grey than in the white matter. There may be many foci, particularly when infective emboli are present, as in ulcerative endocarditis. It may affect the grey matter of several convolutions, particularly in fevers. The part or parts of the brain implicated are nearly always hæmorrhagic and softer than normal. They may be firmer if the hæmorrhage is great. The nerve-cells and fibres are seen in various stages of degeneration and dissolution. Germs may be present or absent. There is proliferation of the connective tissue cells in the walls of the neighbouring blood-vessels, of the neuroglia also to a certain extent, and of the pia mater when the inflammation is near the surface. Fibrous tissue formation takes place around the softened area when it is large, leading to the formation of a cyst, or throughout it when it is small, constituting a fibrosed or sclerosed patch. When pyogenic germs are present, the softened area may go on to form an abscess. In some cases, particularly when hæmorrhage is absent, there may be œdema rather than softening, or changes so subtle as to escape detection except by the finest methods of investigation. Some of the acute intoxications may be of this type, such as some of the food poisonings, the

maniacal forms of exophthalmic goitre, tetanus, diphtheria, &c.

Symptoms.—While they vary considerably, they are generally those which characterise severe brain mischief, viz., headache, delirium, vomiting, somnolence and coma. The earlier symptoms may be irritative and the later paralytic.

Treatment.—Rest in bed, with a free use of purgatives and diuretics should be the initial treatment. Not infrequently bromides, chloral or other sedatives require to be given, and it is often desirable to try the effect of anti-specific remedies.

SUPPURATIVE ENCEPHALITIS—ABSCESS.

Causes.—An abscess is the result of the action of pyogenic germs upon the brain tissue. The affected part may have been in a state of simple encephalitis before the germs reached it, or it may have been healthy. In the latter case there is generally, if not always, disease elsewhere, and hence the abscess is usually to be regarded as a secondary lesion. The germs oftenest found are the staphylococci, streptococci and the diplococcus lanceolatus of Fränkel. The primary lesion which most frequently gives rise to cerebral abscess is disease of the middle ear. It may do so in more than one way—by perforation of the roof of the tympanic cavity, by causing meningitis, septic thrombosis of the lateral sinus, a subdural abscess, or by extending along the lymph spaces or the veins entering the superior petrosal sinus, without implicating the meninges. The inflammation may, in the same way, extend from the mastoid cells, the ethmoid bone, or the frontal sinuses, or even, though very rarely, from the scalp.

When the abscess follows middle ear disease it is likely to be situated in the temporo-sphenoidal lobe, but in the cerebellum when it extends from the mastoid cells.

Another local condition of importance as a cause of cerebral abscess is fracture of the skull, in which case the germs reach the damaged brain either directly, causing first a meningitis, or indirectly through the circulation. The primary condition need not be in the head, for a cerebral abscess may arise when any suppurative or necrotic condition exists in a distant part of the body, or even during the course of a specific fever, *e.g.*, influenza, typhoid. Bone, lung, or liver disease may be mentioned as the most frequent of these distant suppurative conditions in which cerebral abscess occurs, and bronchiectasis is the most important of them all.

Morbid Anatomy.—There is usually only one abscess, which varies in size from that of a pea to that of a large orange. Its contents vary according to its age. In quickly formed cases they consist of ordinary pus mixed up, especially towards the periphery, with reddened and softened brain tissue. There is no definite lining wall, the surrounding brain tissue showing a hæmorrhagic encephalitis of varying extent and degree. In long-standing cases, on the other hand, the pus has a slimy consistence, a characteristic greenish hue (due to the *bacillus pyocyaneus*), and possesses a peculiar odour reminiscent of sulphuretted hydrogen. There is a definite capsule, which in very chronic cases is of considerable thickness. The surrounding brain substance is often normal to all appearance. It is difficult to specify the shortest time required for the formation of a definite abscess wall, but it is probably somewhere about three months. After definite encapsulation has occurred the wall becomes gradually denser and the contents become thicker and even inspissated or calcified. An old abscess may lie dormant and quiescent

for years. It rarely contains any living germs. Such an abscess may light up again as the result of an injury or a fresh infection, and cause a rapidly fatal encephalitis or meningitis. A cerebral abscess, even when deeply seated, may reveal its presence by a meningitis, a thrombosis of a dural sinus, or by a flattening of the convolutions when the calvarium is opened.

Symptoms.—When the symptoms come on acutely after an injury or operation to the head, or in the course of middle ear disease, they are most suggestive. The ruling symptoms are fever, severe headache, mental irritability, delirium, and vomiting. The pain is said to be increased on tapping on the side of the abscess, and MacEwen holds that the percussion note of the skull, which is uniformly dull, becomes more resonant when the lateral ventricles are distended with fluid, as in cerebellar abscess. Drowsiness, slow cerebation, vomiting and optic neuritis are seen in more prolonged cases, but in very chronic cases all suggestive symptoms may be absent. This is particularly the case when the abscess occupies a silent area of the brain. Localisation of the abscess is generally difficult. It becomes feasible when definite convulsions or paralysis are present. In its most common site, viz., the temporal lobe, it may give no localising sign, or it may cause paralysis of the arm, face, or aphasia, if on the left side, by pressure.

Diagnosis.—The history of the case, particularly as to head injury or ear disease, even years ago, is of the greatest importance. Careful examination must be made of the ears, nose, nasopharynx, and the rest of the head for disease, and also of the lungs, &c., for suppurative conditions. The blood may be examined for leucocytosis. In such cases there is little room for doubt when the typical symptoms come on acutely. This is particularly the

case when fever, drowsiness, and perhaps paralysis, supervene in chronic ear disease upon the cessation of the discharge from the ear. The presence of optic neuritis is a help, but it is found in other conditions, such as cerebral tumor, and even in middle ear disease alone.

Prognosis.—It is always grave, but evacuation of the abscess holds out hope in cases in which there is no meningitis or much surrounding encephalitis.

Treatment.—The treatment is surgical. The recognition of an abscess is the first step, and a prompt attempt at evacuating the pus is the second. All that need be said from the medical standpoint is simply that while the abscess is developing, and prior to its recognition, rest in bed, purgatives and diuretics, and the local use of the ice-bag are measures of great importance.

CHRONIC ENCEPHALITIS.

Causes.—Chronic encephalitis may follow upon the acute non-suppurative form, or it may be chronic from the beginning. The same causes may be at work in both these cases. Syphilis and alcohol are said to be common causes.

Morbid Anatomy.—The extent of the change varies greatly. It may involve the entire brain, one hemisphere, a part of a hemisphere, or many limited areas throughout the brain. The affected parts are generally smaller, greyer, and firmer than normal, and may implicate either grey or white matter, or both.

Symptoms.—They vary according to the functions of the part of the brain affected. In some cases this aberration

of function, in others the anatomical position of the lesion, gives the names by which the varieties of chronic encephalitis and myelitis are known.

MYELITIS.

VARIETIES.—The term *poliomyelitis* is applied to inflammation of the grey matter, *leucomyelitis* or simply *myelitis* to that of the white matter. Other terms are used to signify the extent of the inflammation: thus *transverse* means a local inflammation affecting a varying thickness of the cord and extending for a short longitudinal distance only; *diffuse* when it is widely distributed throughout the cord; *meningomyelitis* when both membranes and cord are implicated; *central* when it predominates in the central grey matter around the central canal; *disseminated* when it occurs in multiple foci; *acute* when of sudden onset, and *chronic* when it is insidious.

Causes.—The causes are similar to those of encephalitis, and may be likewise divided into traumatic, toxic and infectious. *Traumatic myelitis*.—This may be caused by a dislocation of the vertebræ, and very rarely by shock without vertebral displacement, but it is much more commonly caused by a tumor, an aneurism, a parasitic cyst, or inflammatory condition of the meninges or of the vertebral bodies. Tubercular disease of the bodies of the vertebræ, commonly called Pott's disease, is the most familiar example. It is usually seen in childhood or youth, though it may occur at any age. The cervical and mid-dorsal regions of the cord are its favourite sites. In dislocation the myelitis is quickly produced, in pressure by a tumor, &c., it is slowly produced. *Infectious myelitis*.—This has been observed during attacks of various in-

fectious fevers, such as rheumatism, typhoid fever, pyæmia, diphtheria, small-pox, gonorrhœa and syphilis. The germs most frequently found in the myelitic foci are the streptococci, staphylococci, pneumococci and the tubercle bacillus. *Toxic myelitis*.—This has been seen in cases of poisoning by strychnine, arsenic, ergotin, alcohol, and in cases of hydrophobia. The myelitis so often seen in cases of pernicious anæmia, leukæmia, chronic nephritis, and other wasting diseases, which shows a decided predilection for the posterior and lateral columns, should probably be grouped under this head. It is thought that in these cases the poison reaches the cord by means of the lymphatics, and hence they are often spoken of as lymphogenic forms of myelitis. It is not to be forgotten that the lack of nutrition characteristic of these wasting diseases may have much to do with the production of the myelitis. In a certain number of cases there is a history of an immediately preceding exposure to cold or over-exertion, suggestive of some causal relationship.

Morbid Anatomy.—The acutely produced lesions are softer, sometimes so much so that the substance of the cord becomes quite diffuent. The colour varies from a distinct red to a pink or dead white. Hæmorrhage is more common in polio- than in leucomyelitis. Later, the colour becomes yellowish if there has been much hæmorrhage, or greyer if not, and the consistency always becomes firmer. Ultimately firm and dense grey areas, rarely cysts, represent the myelitic foci. In other words, there is at first an acute colliquative necrosis, involving a destruction of the nerve cells and the nerve-fibres, followed in time by reactive fibrosis. A suppurative myelitis is rare. Secondary ascending and descending degenerations follow. The distribution of the lesions throughout the cord varies considerably according to the cause. In the polio form the myelitis is limited to that part of the cord injured by the tumor

or the exudate. In many of the cases which follow upon a chill or undue fatigue the myelitis is transverse, *i.e.*, it affects only a short extent of the cord longitudinally, though it may invade it transversely as far in as the central canal. In other cases, *e.g.*, in the majority of the infectious forms, it affects the cord diffusely and over a wide area, implicating either the grey or white matter alone, or both. The disease commonly known as acute anterior poliomyelitis or infantile paralysis is an instance of this variety of acute myelitis. This disease was formerly thought to be an acute inflammation of the cells of the anterior horn. It is now regarded as having a wider distribution. It may in some cases be restricted to the cells of the anterior horn, but in most cases it is general, affecting all the cells of the cord. Its main incidence in individual cases may be either in the cervical or lumbar enlargements, but there is always a tendency for it to invade the whole cord.

Symptoms.—The symptoms of acute myelitis vary according to the character and distribution of the lesions. The simplest way to describe them is to group them under the definite geographical varieties of myelitis, such as compression myelitis, local or transverse myelitis, diffuse myelitis, poliomyelitis. *Compression myelitis.*—The agents producing the pressure have already been referred to. They are caries of the vertebræ, malignant disease, aneurism and parasitic cysts. The caries is due to tubercle in the vast majority of cases, to syphilis in the minority. It causes in many cases marked deformity in the form of an angular curvature of the spinal column, but even though this deformity may be great, it rarely exerts *per se* any actual pressure on the cord, and, conversely, even though there may be no angular curvature or other spinal deformity, there may be great pressure upon the cord. The compressing agent in nearly all cases is a coincident thickening of the meninges and of the cellular tissues around the cord. Though the spinal deformity

does not in itself cause pressure it should always be carefully examined. Tenderness on pressure of the spinous processes of the affected vertebræ is often present, and twisting of the spine causes pain, but pain from pressure on the nerve-roots is rare. Signs of a psoas or a retropharyngeal abscess are to be looked for. In malignant cases there will be most frequently the history of, or the actual presence of a cancer, particularly of the breast, and less frequently of sarcoma of the peritoneum or sacrum or other organ. There may be no deformity of the spine in these cases, but there is frequently agonising pain along the course of the spinal nerves, also acutely painful regions of the skin, which are at the same time anæsthetic to tactile and painful stimuli, a condition spoken of as *anæsthesia dolorosa*. In both aneurism (chiefly aneurisms of the thoracic and abdominal aorta) and tumor cases, pain is common and often severe. The symptoms referable to the cord vary according to the site of the compression. When it involves the thoracic region there may be spasticity of the limbs, due to secondary degeneration of the pyramidal tracts, or there may be paraplegia. These results may appear either before or after the spinal deformity. In many cases numbness, tingling, pins and needles or a girdle sensation first appear, followed gradually by a weakness in the legs, which increases until, in time, it amounts to a paralysis. The deep reflexes are increased owing to the secondary degeneration of the pyramidal tracts. It is only in complete transverse myelitis that they are abolished, and this is rare. Recovery may follow even after the paraplegia has existed for more than a year. When the cervical region of the cord is affected, there may be difficulty in moving the head, narrowing of the palpebral fissure, sinking in of the eyeball (interferences with the cilio-spinal centre), dilatation of the pupils, and unilateral flushing or sweating of the face. In the lumbar region there is also implication of the organic reflexes controlling the bladder and bowels.

Acute localised or transverse myelitis.—The symptoms are similar to those of compression myelitis. They vary according to the site of the lesion. The dorsal region is the commonest. The symptoms are sensory and motor. In the greater number of cases a variety of sensory phenomena appears before any disturbance of motion, but in some, paralysis sets in quickly and soon becomes complete. Numbness, tingling and hyperæsthesia in the legs are the usual sensory disturbances. A girdle sensation is common particularly between the ensiform cartilage and umbilicus. The upper limit of the lesion may be indicated by a zone of hyperæsthesia. Ordinary sensation below this line may be partially or completely lost. The motor symptoms are paralysis and spasticity. The former comes on early, the latter late. There may be at first merely a sense of weight or dragging in the legs, but weakness, passing on quickly to complete paralysis of the legs, soon sets in. The reflexes below the seat of lesion are at first usually abolished and later increased. There is retention of urine and subsequently spastic incontinence, both of urine and fæces. Bed-sores are common. The muscles belonging to the damaged segments of the cord waste and give the reaction of degeneration, those below the lesion do not usually do so, but in course of several months, where the cases last so long, become rigid and spastic, and contractures, particularly about the knee, occur and, along with the involuntary twitchings and spasms of the limbs, cause much discomfort to the patient. When the lesion is in the cervical region of the cord the paralysis involves the arms as well as the legs, and in rare instances the arms only. Hiccough, vomiting, slow pulse, and contraction of the pupil are also characteristic of cervical myelitis.

Acute diffuse myelitis.—The onset is usually sudden, coming on after a chill or during syphilis or one of the infectious fevers. Numbness or tingling, or a girdle sensation in the body or limbs, is not infrequent. The temper-

ature rises at first slightly but soon to a high degree. The pulse is rapid, loss of motion in the lower limbs quickly supervenes and becomes complete. It extends generally to the trunk and arms. Sensation is also lost, though at first there may be hyperæsthesia. The reflexes are lost, though they may at first be increased. The rectum and bladder are paralysed. Bed-sores appear. The muscles waste, and arthritis may develop. The very acute cases die in from five to ten days, others may live for three weeks or even more.

Treatment.—We may discuss the treatment of myelitis under three of its varieties:—*acute*, *chronic* and *compression* myelitis.

Acute Myelitis should be treated by rest, and certainly hot baths have been found of much advantage in arresting the condition at an early stage. The bowels and kidneys should be stimulated, and the patient kept on milk diet. After one or two days hot baths should no longer be used. It is better to induce the patient to lie on his face, so as to permit of the application of an ice-bag to the spine, and also to counteract gravity by raising the spinal cord as high as possible. From the very outset, however, the physician must bear in mind the great risk of bed-sores, and therefore even the ice-bag should only be kept in position for a short time. On no account should counter-irritation be applied to the back, especially below the level of anæsthesia. The skin, wherever it is likely to be pressed upon, must be carefully hardened, and should be kept absolutely clean. Washing with soap and water, and the application of spirit, or of a solution of alum are very valuable, and the skin in the neighbourhood of the anus and urethra should be carefully washed and dusted with boracic powder. Scrupulous cleanliness should be made the guiding rule of the nurses in attendance. It is possible to take off the pressure from the sacrum and from

the heels by means of circular air-cushions, nests made of cotton wool, or other device. There is no disease in which a water-bed, if properly constructed, affords such comfort to the patient, or such assistance to those in attendance upon him.

If there is any specific history, potassium iodide, with or without mercury, should most certainly be administered. The other drug to which reference might be made is ergot. Whether it really arrests hæmorrhage or not is open to question, but in not a few cases its administration has been found of advantage. After the acute stage is over, attention should be paid to the muscles which are paralysed, and massage, sometimes galvanism and sometimes faradism, are of use, but the greatest possible care should be taken to avoid over-stimulation in the early stages.

In *chronic* cases of myelitis warm baths are often of value in relieving contracture and the pains sometimes associated with the disease. Potassium iodide and mercury should always receive attention, and the former should be given a thorough trial. Sometimes counter-irritation to the region of the back is admissible, but due regard to the risk of bed-sores should prevent any rash procedure, and more especially as counter-irritation has not often proved beneficial. Galvanism is the form of electrical treatment most likely to be of benefit, and massage to the affected muscles should be carefully applied.

Compression myelitis is a surgical affection, and certainly in cases where, owing to tubercular disease of the vertebral column, there is pressure on the cord, the treatment is almost purely surgical. Sometimes laminectomy is indicated. In other cases prolonged rest in bed, with extension by means of weights, is preferable, while often the use of poroplastic or plaster jackets is all that is required. The three principles which govern the treatment are rest, extension, and the prevention of rotatory movements. Good food, open air, and such tonics as cod-liver oil, iron,

and so forth, constitute the medical treatment. It is just possible that in certain cases tuberculin may be found not merely of use as a diagnostic agent, but also of value in effecting their cure.

ACUTE POLIOMYELITIS OR INFANTILE PARALYSIS, OR ATROPHIC SPINAL PARALYSIS.

As already stated, the term anterior, which is usually applied to this disease, is misleading, inasmuch as the lesion is rarely limited to the anterior horn-cells, but affects also the cells of the lateral and posterior horns; in short, all the nerve-cells of the cord. From the pathological standpoint, then, its proper place is here among the diffuse lesions of the cord; but inasmuch as all its main symptoms are motor and referable to the lower motor neurone system, most authorities still prefer to place it among the diseases affecting specially the anterior horn-cells.

Etiology.—Age, sex and season all have some influence in the production of the disease. It is commonly met with in the first four years of life, though it may occur in adult or middle age. Boys are more frequently attacked than girls, and more cases occur during the warm than the cold months of the year. It has a feeble tendency to epidemicity, and severals epidemics have been reported. This fact, but still more its rapid onset with fever, suggests that the disease is due to an infection. If it be an infection, which is probable, its nature is still unknown. The infective nature of the disease is in keeping with the fact that babies suffering from chill, fatigue, dentition troubles or an injury, such as a fall, are more likely to be attacked than are perfectly healthy children. In the majority of cases the nerve-cells supplied by the median branches of

the anterior spinal artery are chiefly affected, as if a poison had been brought to them by these branches or their nutrition cut off through an embolic or thrombotic process therein. Both these opinions are supported, but the balance of evidence is in favour of the former.

Morbid Anatomy.—The changes in the cord vary according to their age. The disease is rarely fatal, hence opportunities of observing the early changes do not often occur, but death has occurred, usually from intercurrent disease, in a certain number, at periods varying from one to a few weeks. The following are the changes found in such cases:—A slight leptomeningitis is sometimes seen on one side of the cord in the cervical or lumbar enlargements. A feeling of slight softness may be detected in these enlargements, rarely, in other situations. On section of the cord the grey matter is usually redder than normal in some part of these enlargements, particularly on one side, and may be softer as well. Both sides may be affected, but this is less common than one. The lumbar enlargement suffers oftener than the cervical, but both may be affected. The extent of the affection varies, although it usually extends over more than one segment; but the extent and characters of the changes are best observed under the microscope. The blood-vessels are distended; their lymph sheaths are full of leucocytes; varying-sized hæmorrhages around them are frequent. Thrombosis has been described by some observers as occurring in one or two of the larger arterial branches, but most observers have failed to find any evidence of this. The nerve-cells are strikingly altered. All degrees of change are seen, from a slight swelling and chromatolysis to a complete dissolution and disappearance. Their processes, both dendrites and axones, are similarly affected, being shrunk and beaded in the slighter cases and completely destroyed in the severe ones. Leucocytes often cluster round the

shrunk and degenerated nerve-cells. These changes in the nerve-cells and their processes constitute the chief lesion characteristic of the disease. They are always most marked in the cells of the anterior horns, but also affect those of the lateral and posterior horns. In some cases they are slight, involving only a few of the cells; in others severe, affecting most of the cells at any particular level. Complete destruction of all the nerve-cells at any level is rare, most of the antero-median group of cells presiding over the muscles of the back nearly always escaping. The distribution of the lesions is apparently focal in most cases, and not diffuse all over the cord, *i.e.*, the nerve-cells of one, less commonly of both sides are involved in one or more segments, generally of the lumbar or cervical enlargements, or both, but a careful search discloses a minor degree of change diffusely spread throughout the cells of the whole cord. The other alterations found in the cord are of less importance. They consist in a proliferation of the cells of the neuroglia and of the prolongations of the pia into the cord. The adjacent parts of the white matter of the cord are often affected, involving a destruction of some of the nerve-fibres of the tracts. But any such destruction of nerve-fibres is slight compared with that which occurs in the anterior nerve-roots of the spinal nerves in consequence of the loss of their trophic centres. This secondary degeneration rapidly passes outwards to the periphery, involving the muscles, which quickly atrophy and waste; and not only the muscles but the joints, bones, ligaments and other supporting tissues. On the other hand, in the great majority of cases the patient survives the disease and an opportunity of examining the cord may not arise for months or years afterwards. The cord is then found to be smaller in the part or parts affected. The pia mater is thickened. The nerve-cells in these areas are fewer in number, the extent of the diminution corresponding to the severity of the original attack, some being normal in appearance, others

in various stages of degeneration. Their processes are often stunted. The blood-vessels are distended and their walls thickened. The supporting neuroglia tissue is much denser than normal, the degree of sclerosis being proportionate to the number of nerve-cells which have disappeared. There is generally some sclerosis also in the neighbouring parts of the white tracts.

Symptoms.—The onset is sudden in most cases. A child in good health suddenly suffers from fever and general constitutional disturbance, frequently accompanied by vomiting. Convulsions are rare. These symptoms may be so marked or so slight as to constitute an apparently severe illness or a mere indisposition. The temperature, for instance, may vary from 100°F. to 103°F. Pain is frequently complained of in one or more limbs, particularly when handled, but there is no other sensory disturbance. When these acute symptoms subside, which they may do in a few hours, a loss of power in one or more limbs is noticed. This motor paralysis is the essential feature of the disease. It develops rapidly, reaching its maximum usually within thirty-six hours. In some cases its onset is slower and its full development is not reached for three to five days. It is very variable in its distribution and may affect one or both of the upper or of the lower or all the extremities, or it may affect an arm and a leg on the same or on opposite sides of the body. At first all the muscles of the affected limb seem to be paralysed, but in most cases many of them quickly improve and the paralysis remains only in certain groups of muscles. Muscles which act functionally together are likely to suffer. Thus there is an upper arm type, affecting the deltoid, biceps, brachialis anticus, supinator longus, and a lower arm type, affecting the flexors and extensors of the wrist and fingers. In the legs the tibialis anticus and the extensors suffer more than the glutei and the hamstrings. The muscles of the face and the

sphincters are very rarely involved. The superficial and deep reflexes of the affected limbs are generally absent, the muscles feel soft and flabby to the touch, and give the reaction of degeneration in seven to ten days from the onset of the attack. These changes are not generally noticed for the first few days as the limbs of babies are usually fat and round, but atrophy rapidly supervenes and soon renders the muscles soft and flabby. In the after-history of the case attention may be focussed upon the affected limbs, for the child's general health soon becomes completely restored in the great majority of cases, though a few severe ones end fatally. The paralysis very seldom entirely disappears. It can do so only in very mild cases. On the other hand, it quickly reaches its maximum and then either remains stationary or undergoes some improvement. The degree of improvement is very variable, but it is comparatively slight in many cases. In these, the wasting is extreme. The whole growth of the limb, of its muscles, bones, and ligaments, is retarded, or even arrested, and a *baby arm* or *baby leg* is the result. Subluxation or dislocation of a joint may occur from the relaxation of the ligaments and muscles. Deformities may in time appear from the shortening of unopposed intact muscles.

Diagnosis.—The age of the patient, the sudden onset, the paralysis, rapid wasting and the reaction of degeneration make the diagnosis clear in most cases. In cerebral palsies the paralysed muscles do not waste and do not give the reaction of degeneration, the reflexes are present, and convulsions are common. Multiple neuritis is rare in children, and causes symmetrical paralysis at the periphery of the limbs. Pain is one of its typical symptoms, and other sensory disturbances are prominent.

Prognosis.—Complete recovery is not to be expected. Improvement usually occurs in the paralysed limbs after

a stationary period, and the extent of this impairment, which is often considerable, may be forecast by the degree of completeness of the reaction of degeneration which the different muscles give. There will be no improvement in muscles which give a complete reaction, but it may be great in those which give an incomplete reaction. It is not to be forgotten that contractions and deformities may ultimately arise.

Treatment.—It is of the first importance to keep the child as quiet as possible in bed, and to administer purgatives, diuretics, and, if necessary, febrifuge remedies, because it is upon the arrest of the inflammatory condition in the cord that the future usefulness of the limb or limbs of the child largely depends. The paralysed limb or limbs should be wrapped up in cotton wool, and until the acute stage of the attack has subsided no massage or similar local treatment to muscles should be thought of. Once the temperature is normal, each affected muscle should be carefully and systematically rubbed or otherwise exercised, and much depends on the anatomical knowledge and skill of the masseur or masseuse employed. Galvanism is certainly of benefit, but faradism is only of use where the muscles to which it is being applied respond to the stimulus. There is probably no internal remedy which has much effect in counteracting the toxin, but we favour the administration of ergot, and it certainly should be given a fair trial. *Belladonna* has also been recommended, and certainly cod-liver oil, iron and strychnine will be found of great value in the later treatment.

Surgery has done much to relieve those cases in which, owing to atrophy of certain muscles and contraction of those unantagonised, much deformity has resulted. The procedure found most successful consists in transplantation of tendons, dividing part of the tendons of unaffected muscles, and suturing them to those which are paralysed.

It has been found after some time has elapsed that the results well warrant the troublesome operation. Where it is a lesion limited to certain groups of muscles in a limb, nerve anastomosis will probably yield better results than transplantation of tendons, but the latter procedure is the one most favoured at the present time.

ACUTE AND SUBACUTE POLIOMYELITIS IN ADULTS.

A certain number of cases, apparently exactly similar to those of children, have occurred in adults, and they are thought by many to be true cases of poliomyelitis. It is probable that there are such cases, but many of the cases so described have been cases of multiple neuritis, which it is very difficult in adults to differentiate from acute poliomyelitis.

LANDRY'S PARALYSIS—ACUTE ASCENDING PARALYSIS.

Etiology.—The cause is obscure. There is considerable evidence in support of the disease being caused by a toxin which rapidly destroys the lower motor neurones. The nature of this toxin is not known, but it seems probable that it is not always the same. In one case it may follow typhoid fever, in another diphtheria, in another some pyogenic process, in another syphilis, and so on. The disease occurs most commonly in adult males.

Morbid Anatomy.—Landry found no change either in the central or peripheral nervous system, and several recent writers support him. Some observers have described more or less extensive changes in the motor cells of the

cord, and others (the most numerous) an extensive destructive and hæmorrhagic parenchymatous inflammation in the peripheral nerves. There are other signs of a toxin circulating in the system, in the enlarged and congested spleen, in the granular degeneration of the kidneys and liver, and in profuse sweating which is often present.

Symptoms.—Muscular weakness in the legs sets in suddenly and without warning, save a little tingling in some cases. It gets rapidly worse until it becomes a complete paralysis. This may happen within a few hours. The paralysis spreads to the trunk and then the arms, in which it may be complete in a few days time. But it does not stop here. It quickly advances upwards, paralysing in turn the neck muscles and the muscles of deglutition, speech and respiration. The muscles do not waste, nor do they give the reaction of degeneration. The reflexes are lost, but sensation is usually intact. The sphincters are not involved and the organic reflexes—the trophic, vasomotor and mental functions—remain normal. There is no fever.

Diagnosis.—The only disease which may be confused with it is multiple neuritis, and then only when marked sensory symptoms are present. In such cases it may not be possible to make the distinction.

Prognosis.—Most cases end fatally in from two days to two weeks. Death is due to paralysis of the vital centres in the medulla. A few cases live longer, and some few cases may even recover.

Treatment:—Until we are able to define the extent and the domain of the nervous system implicated in this disease, as well as the toxin responsible, no treatment can really be satisfactory. Within the last few months we have seen a

case of the disease rapidly advancing towards death, and only arrested when 20 minims of ergot were administered thrice daily. What the exact effect of the ergot was, it is impossible to say. It succeeded after potassium iodide and every other remedy had failed, and certainly in cases where Landry's paralysis is threatening the patient's life the drug is well worth a trial.

CHRONIC MYELITIS.

Etiology.—Many inflammations and degenerations of the cord, as in the brain, are of insidious onset. They may follow gradually upon the acute or subacute inflammations, or they may arise independently. In either case they are of slow development and progress. The causes are the same as in the acute diseases. Special mention must be made of syphilis and tubercle, which each cause a specific form of myelitis.

Morbid Anatomy.—The change may be in the white or the grey matter, or both. It may be focal, scattered or diffuse in distribution. Its characters depend upon its age. Chromatolysis, pigmentation, atrophy and disappearance of nerve-cells, fatty degeneration and disappearance of nerve-fibres, and increase in neuroglia tissue occur. The overgrowth in neuroglia is largely compensatory: it is very slow, and shows after two or three months a loose spongy tissue whose spaces are filled with fluid, or scavenger cells containing fat and pigment. At this stage it has a gelatinous appearance to the naked eye. It becomes gradually denser and more extensive, so that in about ten to twelve months it forms a dense, fine, fibrillar tissue in which a few scavenger cells may still be seen. The walls of the blood-vessels are thickened. It is of a grey colour, dry and firm, and causes considerable shrinking of that part of the cord

which it implicates. The term sclerosis is generally applied to all such chronic hardenings in the cord and brain. Firm fibrous bands, similar to those seen in other organs, do not occur in the central nervous system, except in the pia mater and its invading processes, and around blood-vessels. It is present in extensive destructive lesions, but it always originates in ingrowths from the pia mater. A diffuse increase of the neuroglia tissue, *i.e.*, a gliosis of the cord, as in the brain, is probably congenital in origin. When the foci of sclerosis are scattered throughout the cord and brain they constitute the disease known as disseminated or insular sclerosis. When they are limited to definite parts of the cord and brain, *e.g.*, the motor or the sensory domains, they constitute the system diseases of the cord and brain. Some of those diseases affect the system of the first motor neurone, some that of the second, some both, some that of the sensory neurone, and others a combination of these systems. They constitute some of the most important and best known among the nervous diseases and require careful consideration.

DISEASES OF THE WHOLE MOTOR SYSTEM— FIRST AND SECOND NEURONES.

The various muscular atrophies and dystrophies may be grouped here with convenience, if not yet with scientific justification, under the three heads of—(1) Progressive muscular atrophy (central); (2) peroneal progressive muscular atrophy (neural); (3) muscular dystrophies.

CHRONIC ANTERIOR POLIOMYELITIS — PRO- GRESSIVE MUSCULAR ATROPHY.

Varieties.—(1) Spinal muscular atrophy; (2) amyotrophic lateral sclerosis; (3) bulbar paralysis.

Etiology.—The disease is of insidious onset and occurs chiefly in adults. Its cause is obscure, but analogy suggests that it is due to the slow action of some poison of metabolic or infectious origin. There is a history of syphilis in many cases, and it is believed to have some causative influence. In other cases there is a history of exposure to cold, fatigue, overwork, of mental worry, or even of injury. Heredity does not seem to play an important part, though several members of a family may be attacked, particularly by the rare infantile form.

Morbid Anatomy.—The principal lesion is a degeneration and atrophy in the motor cells of the central nervous system. In many cases it is restricted to the cells of the anterior cornua of the cord or extends slightly to the cells of Clarke's vesicular column. This type is sometimes known as the *Aran-Duchenne* type of progressive muscular atrophy. In other cases it extends to the motor cells in the *medulla and pons*, and in a few even to the *cortex*, affecting the motor cells in the *ascending frontal convolution*. In still other cases there is sclerosis of the *crossed pyramidal tract* or *anterolateral ground bundles*, or both, associated with the degeneration and atrophy of the motor cells of the anterior cornua, a combination which is known as *amyotrophic lateral sclerosis*. When it attacks the motor cells of the medulla first, or early, it is known as *bulbar paralysis*. All these types are merely variations of the one disease, viz., a degeneration of the whole system of the efferent or motor neurone. There is a progressive diminution in number of the motor nerve-cells, and many of those still persisting show atrophy and degeneration. This change generally begins in the cervical region of the cord in the *Aran-Duchenne* type and spreads gradually to the thoracic and lumbar regions. An involvement of the medulla and pons generally occurs late, except in the bulbar type, where it is first or early, descending later to the cord. It is only in

rare cases that the disease is confined either to the medulla or to the cord. The place of the lost nerve-cells is taken by a new formation of neuroglia tissue, which contracts as it gets denser and older, so that a diminution in the size of the affected anterior cornua becomes marked in old cases, though it may be slight in the earlier stages. Combined with the change in the motor nerve-cells there is a corresponding atrophy and disappearance of their axones, and therefore of the anterior root bundles, both inside and outside the cord. The morbid change descends in the spinal nerves to the terminations in the muscles, which are correspondingly degenerated and atrophied. The same thing is seen in the ganglionic cells of the cerebral motor nerves and in the muscles they supply. In the amyotrophic lateral sclerosis type there is a sclerosis in the crossed pyramidal tracts or in the ventro-lateral ground bundle (the direct cerebellar and antero-lateral ascending tract of Gowers escape), associated with the Aran Duchenne type of changes in the lower motor neurone. The sclerosis is apparently primary, hence the muscles are spastic and not flaccid as in the Aran-Duchenne type. Pure cases of either type are rare, whilst mixtures of the two are common.

Symptoms.—Rheumatic pains are frequently complained of before any muscular wasting is noticed, but sooner or later this makes its appearance. It is different in character and degree in the Aran-Duchenne and amyotrophic lateral sclerotic types, and it is therefore convenient to describe it separately in each, though it is to be remembered that typical cases of either are rare compared with cases showing some degree of mixture of the types. In the Aran-Duchenne type it usually begins in the muscles of the ball of the thumb and of the little finger of one hand. They waste, causing the thenar and hypothenar eminencies to disappear. The interossei and lumbrical muscles atrophy in turn, and

therè is in consequencè a loss of the faculty of performing delicate manipulations. Ultimately the contraction of the flexors and extensors, unresisted by the atrophied muscles, produces a claw-like hand, called the "main en griffe" or griffin's hand. The atrophy extends to the muscles of the arm, the flexors usually before the extensors. The deltoid and biceps suffer early. The lower two-thirds of the trapezius and certain of the scapular muscles are next attacked. The upper third of the trapezius and platysma myoides do not atrophy. It may be long before the atrophy extends to the muscles of the trunk, and the arm and trunk muscles may be much attenuated before the muscles of the legs begin to succumb. The face and some of the neck muscles are affected late if at all. Ultimately the intercostal and abdominal muscles also waste, and the patient may present the appearance of a living skeleton. He may still be able to walk about, but lordosis and other contractions almost always come on. In all the muscles affected, and in some not yet affected, fibrillary twitching is common. There is electrical irritability, the galvanic persisting for the longer time, and there may be a partial reaction of degeneration. The tendon reflexes are gradually lost. The organic reflexes are normal, and though the patient may complain of numbness and coldness, sensation is unimpaired. The muscles are flaccid or "atonic," and the degree of paralysis or loss of power is usually proportionate to the wasting. In the amyotrophic lateral sclerosis type, spasticity precedes the wasting, which is never so extreme as in the other type. Moreover, the degree of paralysis or loss of power is often out of proportion to the wasting. This "tonic" atrophy first involves the arms and then the legs. The reflexes are increased. The organic reflexes may be normal, but sexual power may be lost early. In both types and their combinations the disease may last for years, and even after it involves the lips and the speech the patient may live for

a long time without symptoms occurring which threaten life. In the final stages the memory fails and a condition of dementia may come on.

Diagnosis.—In the early stages it may be mistaken for paralysis of the ulnar nerve, but the interossei do not suffer in the latter. Siringomyelia may simulate the spastic type of muscular atrophy, and while it is easy to distinguish them when the sensory symptoms of the former are well-marked, it may be impossible to do so when they are not.

Prognosis.—The disease is generally steadily and surely progressive towards a fatal issue. Its progress is slow, and temporary arrests of long duration may occur, but it will ultimately be certainly fatal.

Treatment.—The treatment of this condition is unsatisfactory in as much as we are still ignorant of the toxin concerned, and also of its mode of attack on the nerve-cells of the anterior horn. Hypodermics of strychnine injected deeply into the affected muscles have been found to give good results if the treatment is continued for a long time. The dose is $\frac{1}{100}$ th to $\frac{1}{50}$ th of a grain. Electricity and massage are both useful, and an attempt should be made to prevent deformity, sometimes by recourse to surgical procedures. Feed the patient well, and try the effect of tonics, such as arsenic, iron, &c.

BULBAR PARALYSIS—GLOSSO-LABIO- LARYNGEAL PARALYSIS.

This is, as already mentioned, merely a variety of progressive muscular atrophy, in which the nerve-centres, which suffer early and chiefly, lie in the medulla. Some of those of the pons may also suffer. The paralysis therefore

affects the lips, cheeks, tongue, throat and larynx, interfering with speech, including phonation, and with mastication and swallowing. It may run an acute or chronic course.

The Acute Form.—Certain cases come on after influenza or diphtheria or some other continued fever, and some occur in the victims of syphilis. In others there is no definite indication of the nature of the poison. The lesions have a correspondingly acute character. Thus the nerve-cells of the pons and medulla, and sometimes of cerebral nerves higher up, *e.g.*, the third and fourth nerves, show acute degenerative changes accompanied by hæmorrhages or foci of acute softening. Emboli and endarteritis may be present in such cases, and it is difficult to say how far the patches of softening, &c., are due to diminished nutrition, and how far to the direct action of a poison. There is a strong similarity between such cases and many cases of acute poliomyelitis. The symptoms set in very suddenly, and hence this form of bulbar paralysis is often described as being apoplecticiform. There may be headache, vomiting, and unconsciousness. There is paralysis of some of the eye muscles when the higher nuclei are implicated, frequently nystagmus, and a cerebellar gait. In addition to the glosso-labio-laryngeal paralysis there may be a monoplegia, an ordinary or a crossed hemiplegia, or a paralysis of all four limbs. The disease soon terminates fatally.

The Chronic Form.—In this form, as in progressive muscular atrophy generally, there is usually no definite evidence of the nature of the poison which is believed to be the cause, though sometimes there is a history of syphilis or lead poisoning or worry. The lesions are, as already mentioned, of a chronic character. The symptoms begin insidiously. A slight defect or indistinctness in speech, particularly in pronouncing the dentals and linguals, is usually first noticed. The lingual and palate muscles

become weak, and fluids may regurgitate through the nose, though the palate muscles are often much later in being affected. Fibrillary tremors are seen in the muscles of the lips, cheeks and tongue. Mastication is performed fairly well, but owing to the weakness of the cheek muscles, and of the tongue, the bolus of food is pushed back to the pharynx with difficulty, and some of the food is apt to remain between the jaws and the cheek. The saliva is increased. It tends to accumulate in, and dribble from the angles of the mouth. The pharyngeal and œsophageal muscles become involved. Regurgitation into the nose becomes more pronounced, and food may enter the larynx, either causing fits of coughing, by which it is expelled, or setting up an aspiration pneumonia on reaching the lungs. Paralysis of the muscles of the larynx is rarely so great as that of the lips and tongue, and by the time the patient is aphonic he can rarely put out his tongue at all, and his lower lip is thick and hangs down, allowing of the saliva to trickle out more than ever. Though the expression of the face becomes so listless and apathetic, the mental power remains unimpaired. The electrical excitability of the muscles is diminished, and there may be a partial reaction of degeneration. There is no change in sensation. The disease spreads in time downwards in the cord, affecting the cells of the anterior horn, and often also the lateral tracts.

Diagnosis.—The early incidence and prominence of the paralysis in the lips, tongue and throat make the diagnosis easy. A similar condition may arise from a unilateral (rarely) or bilateral lesion in the lower part of the ascending frontal convolution or from a destructive lesion about the knee of the internal capsule. This condition, which is rare, is spoken of as pseudo-bulbar paralysis or bulbar paralysis of cerebral origin.

Prognosis.—The disease progresses steadily downwards until death, which may be due to choking, to failure of the

cardiac or respiratory centres, to an aspiration pneumonia, or some other complication.

Treatment.—The remarks already made in describing the treatment of the preceding disease (progressive muscular atrophy) are applicable here. Rest, freedom from excitement, good feeding, the use of electricity, and especially galvanism, are amongst the indications we should follow. Strychnine may be used hypodermically and arsenic given internally. If syphilis is suspected, potassium iodide should be administered, and the treatment kept up for a considerable time. Where the laryngeal muscles are paralysed, the patient must be fed by a stomach tube to obviate the risk of food entering the bronchial tubes.

PROGRESSIVE NEURAL MUSCULAR ATROPHY.

—THE PERONEAL TYPE.

This type of atrophy was described by Charcot, Marie and Tooth. It shows certain differences from the central type. In the first place, it usually begins in early childhood as a hereditary or family affection, affecting the peronei, anterior tibial and feet muscles. A double club-foot is the result. It is generally years before it extends to the arms or body, though in rare instances it may begin in the hands before affecting the legs and feet. In its extension from the legs to the arms it generally attacks the small muscles of the hand before the other muscles of the upper limb. Fibrillary twitchings occur in the affected muscles, which also show a loss of electrical excitability. Sensory disturbances are frequently present, which is in marked contrast to all other forms of muscular atrophy.

THE MUSCULAR DYSTROPHIES.

Under this heading are included atrophies of muscle apparently dependent upon primary changes within the muscles themselves, and not upon changes within the central nervous system, which have rarely been observed to occur in these cases. It would therefore be more correct to describe them under the diseases of muscles, but it is, on the other hand, more useful for clinical purposes to describe them here and to leave the question of their proper category for future determination. Many clinical types have been described, based upon the age of onset, the muscles first affected, the presence or absence of hypertrophy. Erb says they can all be grouped in two classes—(1) those occurring in childhood; (2) those occurring in youth or adult life. In the first group the muscles may be either apparently hypertrophied or atrophied. The hypertrophied includes the pseudo-hypertrophic paralysis of authors and other similar cases with real hypertrophy, whilst the atrophied includes the facio-scapulo-humeral type described by Landouzy and Dejerine, and a similar form in which the face is not involved. In the second group, on the other hand, the muscles are always atrophied. It constitutes the juvenile type of Erb, and is similar in every respect to the pseudo-hypertrophic paralysis, except that in it there is never any enlargement of muscles, and the age is older. It will suffice to describe in detail the pseudo-hypertrophic form.

PSEUDO-HYPERTROPHIC PARALYSIS.

Etiology.—It is a disease of childhood, rarely beginning as late as fifteen years of age. Heredity plays an important part in its incidence. The disease may occur in two or more generations and attack several members of the same family. Males are more frequently affected. The disease is usually transmitted through the mother, though she herself generally escapes,

Morbid Anatomy.—The individual muscle-fibres of the affected muscles split longitudinally. Some of the fibres hypertrophy, but most have their nuclei increased. The interstitial tissue between the fibres is increased. The muscle-fibres become vacuolated, fissured and atrophied and their nuclei become correspondingly increased, with the production in places of appearances which suggest an attempt at reformation of muscular fibres. Fat may be deposited in the interstitial tissue in large amount, and it is this local lipomatosis which confers upon the muscles their hypertrophied appearance and upon the disease the name of pseudo-hypertrophy. No lesion has been found within the nerves or in the central nervous system in the great majority of cases, though in a few atrophy of the ganglion cells of the anterior horn has been described.

Symptoms.—An enlargement of the calf muscles not accompanied by an increase but by a decrease in muscular strength is noticed. Movements are more clumsy, and the climbing of stairs more difficult. The extensor muscles of the legs, the glutei, the lumbar muscles, the deltoid, and infra-spinatus become in turn similarly affected. The muscles of the forearm, face and neck rarely suffer, whilst others waste, such as the latissimus dorsi, teres major, and the lower part of the pectoralis major. The biceps generally wastes and the triceps usually hypertrophies, but the opposite may occur. As a whole, the leg muscles are more affected than those of the arm, *i.e.*, the upper arm, for, as already mentioned, the hand and forearm escape. The patient stands with the legs apart and finds great difficulty in bringing the legs together. The spine is curved and the abdomen protruded. One of the most characteristic signs of the disease, as shown by Gowers, is the way the patient acts in assuming the erect posture after being laid upon his back. He turns round on his face, gets on all fours, pushes his body upwards with his hands, which he brings along

the floor towards his feet, then raises one side of his body by placing one hand on one knee, then the other side with the other hand on the other knee, and gradually climbing up his thighs pushes his body into the erect posture. Wasting proceeds gradually until in time the muscles appear smaller than normal. Lordosis, club-foot and other distortions and contractions occur. Owing to the weakness of the muscles of the shoulder girdles, the shoulder blades are prominent and the shoulders loose. If the hands be placed under the arms and an attempt be thus made to lift the patient, the shoulders are raised to the level of his ears so as almost to suggest his slipping through. The knee jerks are gradually lost. The reaction of degeneration is rarely given, but the electrical excitability to faradism and galvanism is diminished.

Diagnosis.—The age of the patient, the appearance of the muscles, the gait and movements generally enable a diagnosis to be made with ease.

Prognosis.—The disease is slowly but surely progressive until the patient becomes bed-ridden. Death generally occurs from some intercurrent disease.

A few words may be added descriptive of the facio-scapulo-humerical type in which the face is involved. The zygomatic muscles are specially affected. There is loss of the naso-labial fold, projection of the lower lip, and inability to whistle or pronounce labials.

Treatment.—Attend to the general health, and endeavour to counteract deformity by performing tenotomy. No medicinal agents have so far been found of any real benefit other than the use of tonics should the patient's health appear to require them.

DISEASES OF THE UPPER MOTOR NEURONE.

It is doubtful if there is any disease whose lesions are entirely restricted to the upper motor neurone, but there are a few thus restricted, clinically at any rate, for the greater part of their course, and they may with convenience be grouped under this head. The most important members are infantile spastic paralysis, primary lateral sclerosis, and secondary lateral sclerosis.

INFANTILE SPASTIC PARALYSIS—SPASTIC OR INFANTILE DIPLEGIA—BIRTH PALSIES —LITTLE'S DISEASE.

Etiology.—Injury at birth is the cause of the majority of the cases. Hence they are commonest in first labours or forceps cases. Premature birth before the development of the pyramid tracts is believed to be the cause in a few cases. A foetal meningo-encephalitis, due to syphilis, may be the cause in some others.

Morbid Anatomy. — Meningeal hæmorrhage is the commonest result of the injury. It may come from the veins or the longitudinal sinus or an actual tear of the meninges. It is usually extensive, involving both sides, and by its pressure causes atrophy and sclerosis of the underlying cerebral cortex. Thus the motor centres suffer, those for the legs usually more than the arms, and in more extensive hæmorrhages the frontal lobes also, with a consequent disturbance of the mental condition, resulting in varying degrees of imbecility and idiocy. If the hæmorrhage and destruction is localised a porencephaly may result. In syphilitic sclerosis the affected convolutions may be much smaller than their neighbours,

Symptoms.—The symptoms may be present at birth, but it is more usual for them to appear during infancy. Convulsions are common. Weakness and stiffness in the arms and legs are still more common. The legs are most affected and the arms may, in certain cases, suffer so slightly that a paraplegia and not a diplegia seems to be present. The child is unable to walk, or in some cases even to sit up, long after the age at which he should be able to do both. When the arms are markedly affected he is unable to grasp an object easily, and all voluntary actions of the hands may be accompanied by stiff, spasmodic jerking movements, a condition to which the name "*chorea spastica*" has been given. A *bilateral athetosis* may also be present. The face muscles may twitch, and the head be turned from side to side. Various degrees of mental deficiency are common.

Prognosis.—All cases if left alone will run an unfavourable course, though some of the syphilitic cases may reach an adult age. Early operative procedure in the hæmorrhagic cases may do good.

Treatment.—There is no question of the immense value of prompt operative interference where a meningeal hæmorrhage has occurred at birth, and the success obtained in the cases already submitted to operation warrants an attempt being made to clear out the blood clot in every case. Should the patient be seen long after the hæmorrhage has occurred, little can be done to restore the damaged brain tissue. It is possible that potassium iodide may be of some service in a small proportion of cases, but generally speaking the benefit obtained from good feeding and from educational methods is extremely limited.

PRIMARY SPASTIC PARALYSIS OF ADULTS—
PRIMARY LATERAL SCLEROSIS—PRIMARY
SPASTIC PARAPLEGIA.

Etiology and Morbid Anatomy.—It is assumed at the present time that this disease exists as a separate entity. It has been ascribed to syphilis and influenza, and it is supposed that it consists in a sclerosis of the pyramidal tracts, beginning below and progressing upwards. Many of the reported cases have turned out afterwards to be early stages of an insular sclerosis. Others may have been really secondary to a lesion higher up in the tract, though apparently primary, and still others have developed later into general paralysis. Increasing doubt is therefore thrown upon the separate existence of the disease.

Symptoms.—The legs almost always suffer first, and it is rare for the arms to be affected at the same time as the legs. Weakness in the legs is the chief complaint, and the patient soon experiences a tired feeling. Stiffness and not weakness may be complained of, and in any case, after a few months, both weakness and stiffness are generally present, so that the patient moves with a stiff hesitating gait and is apt to trip over any irregularities in the ground. This gait is most marked in the morning, at least for a time. The rigidity may be most pronounced when the legs are extended. Thus when the flexed leg is partially extended, the muscles complete the extension in a sudden and spasmodic fashion. This action is sometimes likened to that of a *clasp knife*. The adductors are also affected, in consequence of which the knees are kept close together and the legs even crossed in walking. In these cases the legs are separated with considerable difficulty. The knee jerk is exaggerated and ankle clonus is easily elicited. Babin-ski's sign is positive. The superficial reflexes are increased.

Towards the final stages the organic reflexes may fail and contractures may occur in the affected limbs. The muscles themselves do not waste till late in the disease, and even then not always. The arms are not usually affected for years after the legs. There are no sensory symptoms. The course of the disease is steadily downwards as a rule, though it generally lasts for years and there may be long periods of apparent quiescence or even of improvement.

Treatment.—Exercises and massage for the affected muscles are helpful in combating the tendency to increasing spasticity. Hot baths are not merely comforting to the patient, but appear also to afford temporary, if not permanent benefit. Where there is the remotest suspicion of syphilis, the usual antispecific treatment should be carried out.

SECONDARY SPASTIC PARALYSIS.

Etiology and Morbid Anatomy.—Any destructive lesion of any part of the pyramidal fibres will result in a descending degeneration of those fibres below the site of the lesion, gradually extending downwards as far as their terminal arborisations around the cells of the anterior horns of the cord. Cerebral hæmorrhage involving the internal capsule is the commonest of these lesions, but tumors, meningo-myelitis, fracture and caries of the bodies of the vertebræ and insular sclerosis may likewise cause it.

Symptoms.—They are identical with those of the so-called primary form.

There are other forms of spinal disease, such as Erb's syphilitic spinal paralysis and Sach's amaurotic family idiocy, which may be regarded as diseases of the upper motor neurone. The great tendency for mimicry of spastic paraplegia in hysteria should also be mentioned. Readers are

referred to special treatises for a description of these conditions.

Treatment.—The same treatment should be followed in this condition as suggested for the primary form of spastic paralysis. The reader is also referred to the remarks made under the treatment of Cerebral Hæmorrhage (page 878).

DISEASES OF THE LOWER MOTOR NEURONES.

The same thing may be said of the lower as of the upper motor neurones, viz., that it is extremely doubtful if any nervous disease is restricted to them, but certain forms are so restricted for a time at least clinically. Both acute (infantile paralysis) and chronic (progressive muscular atrophy) poliomyelitis may in certain examples illustrate this statement for the greater or even the whole part of their course, but their more correct scientific position is under diseases of the whole motor neurone, in which they have been placed.

DISEASES OF THE SYSTEM OF SENSORY NEURONES.

The chief of these diseases is *Tabes Dorsalis*, and it is the only one which undeniably belongs to this group. *Herpes Zoster* and *General Paralysis of the Insane* are also placed here by some authorities.

TABES DORSALIS—LOCOMOTOR ATAXIA— POSTERIOR SPINAL SCLEROSIS.

DEFINITION—A disease of the exogenous fibres of the posterior roots of the spinal nerves, of the posterior columns of the cord, and at times of cerebral and spinal ganglia and of endogenous fibres and peripheral nerves, characterised by shooting pains and other sensory disturbances, marked inco-ordination, loss of knee-jerks, trophic changes and optic atrophy.

Etiology.—Syphilis is blamed for the great majority if not for all cases. Injury, over-exertion, exposure and sexual excesses are thought by many to have a causal influence. It has been suggested that the excessive use of certain cord tracts, which had been previously weakened by syphilis or the strain of life or even by developmental deficiency, may determine the onset of the disease. It is much more common in males, nearly ten to one, and in those of adult life, most cases occurring between the thirtieth and fiftieth years.

Morbid Anatomy.—The nervous lesions may be grouped under three heads:—(1) in the extra-medullary parts of the spinal nerves, (2) in the spinal cord, (3) in the brain. The extra-medullary portions of the nerves have been shown to be diseased in comparatively few cases, degenerations having been found in the nerve terminations in the skin, in the peripheral nerves themselves, in the spinal ganglia, and lastly in the sensory roots between the ganglia and the cord, the last two situations being the most frequent. These extra-medullary nerve lesions, even when present, have occurred late in the disease and are generally thought to be of secondary importance. The spinal cord lesions, on the other hand, form the most striking and important part of the morbid changes characteristic of the disease. The region of the cord affected is, in most cases, the lumbar enlargement, from which it spreads upwards, but

in a certain number of cases it primarily affects the dorsal or cervical regions. The lesion begins not in the cord itself, but in the intra-medullary parts of the roots of the spinal nerves, *i.e.*, in the nerve-roots which come from the intervertebral ganglia. According to Orr and Row, the exact part of these roots where the lesion begins is the place where the fibres lose their neurilemma just outside their passage through the pia mater into the cord. The causal poison being carried upwards in the lymphatics of the nerve-sheaths causes degeneration to begin in the myelin coats at this point, and to extend slowly inwards and upwards along the course of the nerve-fibres in the cord to such a distance as the virulence of the toxin and its duration is able to prove effective. Moreover, it is probably not all of the fibres, but only some of them, which are degenerated. These exogenous root-fibres enter the cord in two bundles, the one forming the short fibres of Lissauer's tract and the other the columns of Burdach. Many authorities maintain that Lissauer's tract is greatly affected in the early stages of tabes, whilst Mott, Orr and Row say that if affected at all it is only slight. Burdach's column is prominently affected, and since the fibres of each spinal nerve entering the cord in succession from below upwards do so between the posterior roots and the nerve-fibres which have entered lower down, the affected fibres in the lumbar region will at first occupy an outer part of Burdach's column, then more of it as affected fibres successively enter the cord, until a large part of it may be involved. The extent of the degeneration, even in the affected parts, is never so complete as in pressure myelitis, numerous healthy fibres being seen scattered among the degenerated ones. But when segments are reached in which only healthy fibres enter the cord, the outer part of Burdach's column will contain healthy fibres, while the rest of it and the outer part of Goll's column will be degenerated. These degenerated fibres will be in this way pushed successively

nearer and nearer to the middle line, until in time they occupy only Goll's column. And further, since newly-entered fibres lie not only close to the posterior horn but near to the posterior commissure, the anterior parts of Burdach's column are less affected than the middle and posterior, and the anterior parts of Goll (the anterior root zone of Flechsig) also escape in the upper parts of the cord. This is the usual course shown by the degenerations in a typical case of tabes, but the degree in which the different tracts are involved at different levels varies greatly in different cases. When the disease begins in the cervical or dorsal regions of the cord the degeneration is prominent in the posterior roots of these regions and in the columns of Burdach, generally throughout the whole length of the cord. There are still other lesions in the cord in many cases. A degeneration in the direct cerebellar tract passing upwards to the medulla is common, and some degeneration also in Gowers' anterior lateral ascending tract may be present. This ascending degeneration is believed to be due to atrophic changes in the cells of Clarke's vesicular column. In advanced cases the cells of the anterior cornua may be involved, resulting in muscular atrophy, but muscular atrophy is not seen as a rule till late in the disease. Much of it is due to disuse, but in late stages it is partly due to changes in the anterior horn-cells. Charcot's joint affection and other trophic changes may be due to this implication of these cells, but they may occur quite early in the disease. The brain changes are variable. Optic atrophy is the commonest (it is frequent), and degenerations may occur in other cranial nerves, *e.g.*, the third, fourth, sixth, and particularly the fifth. Mott has described changes in the pyramidal fibres believed to be due to atrophy of the cortical cells, which bring the disease into line with general paralysis. The nature of the morbid lesions of tabes may be summed up as follows:—It is a primary degeneration of the myelin sheaths of the

posterior nerve-roots and their extensions into the cord and brain, the axis-cylinders and certain nerve-cells suffering later. The neuroglia tissue increases generally as the degeneration progresses, *i.e.*, it undergoes a compensatory hyperplasia which will prevent any shrinking of the nerves and columns of the cord if it keeps pace with the primary myelin degeneration, but allow of a certain amount of shrinking if it does not do so, which is usually the case. A varying degree of sclerosis of secondary formation is thus seen in most cases.

Symptoms.—The disease generally begins slowly, and runs a very chronic course. Its symptoms comprise disturbances of ordinary sensation, of the reflexes, of motion, of the special senses, of the visceral reflexes, of the vaso-motor and trophic functions, and sometimes of those of the brain. Some of the symptoms, particularly sensory and reflex ones, may be present for a long time before any motor symptoms causing ataxia appear, and this period is sometimes called by clinicians the *pre-ataxic stage*. The motor symptoms consist in various inco-ordinations of muscular movements without any wasting or weakness of the muscles. This *ataxic* stage is the prominent one throughout the disease. It may last for years. It may be followed by a final stage, which may also last for years, of muscular weakness or *paralysis*, in which the patient is bed-ridden. The symptoms require detailed description.

The sensory symptoms.—*Lightning* or *shooting pains* may be the earliest symptoms. They are of a sharp stabbing character, lasting for a second or two or longer. They may be present for hours or days in one position, recurring at irregular intervals. They are commonest in the legs, and have been mistaken for sciatica when confined to one leg. They often flit from one leg to the other or to another part of the body, and are apt to come on during cold or damp weather or after fatigue. A *girdle*

sensation or feeling of a tight belt around the body may be present. There may be *anæsthesia* over areas of the feet and legs, and even when sensations are appreciated their conduction is delayed, thus retardation of the sense of pain is common, a pin prick being felt at once, as a touch, and later (a second or two, or even ten) as a painful sensation. This painful sensation may persist longer than usual. There may be *paræsthesia* or abnormal sensations of different kinds, *e.g.*, of burning, tingling, pricking. The patient may complain of a feeling in the soles of the feet as if cotton was interposed between the skin and the floor when walking with bare feet. In a few cases there is *polyæsthesia*, *i.e.*, one touch is felt as many. In others there is *allocheiria*, *i.e.*, a touch on one place is felt on the corresponding spot on the opposite side of the body, and not where the touch was made. The *muscular sense* is usually affected early, and becomes much impaired, though it may remain intact. It may be tested by tying bags containing different weights tightly to the limbs or asking the patient to shut his eyes and then recognise the position in which his leg is placed.

The *reflex symptoms*.—The deep reflexes, *viz.*, the knee and ankle jerks, are lost, and when the arms are affected the wrist and bicep jerks are also lost. This loss of knee and ankle jerks is one of the earliest and most important symptoms of the disease. It may be present for years before ataxia. The loss may be unequal in the two legs, and if not complete at first, tends to become so later. (The knee jerks are rarely absent in healthy individuals). The skin reflexes in the legs are usually impaired.

The *motor symptoms*.—The ataxia develops gradually. It is thought to be due to a disturbance of the afferent impulses from the muscles, joints, and deep tissues. It may be tested by asking the patient to stand erect with his feet together and his eyes shut. He sways and may fall (Romberg's symptom). This inability to maintain

his equilibrium when his eyes are shut may be noticed by the patient on washing his face, and may be his first indication that something is wrong. It may be noticed in other ways. He can ascend more easily than descend steps. He is likely to sway when turning rapidly. His gait soon becomes characteristic. It is best brought out by making the patient walk without boots or shoes. He stands with his feet wide apart so as to broaden his base of support, and in walking he throws out his legs violently, raising his feet too high, and brings them down heel first with a thump, or the whole sole comes flat on the floor. The nutrition of the muscles is good. There is no atrophy or loss of power, but he needs a stick in walking to steady himself with. As the ataxia increases he needs in time two sticks. When the arms are affected, he may perform delicate movements clumsily, such as buttoning his collar. He may be unable to touch the tip of his nose with his forefinger when asked to do so with his eyes shut, or to bring the tips of the fingers into apposition when the arms are outstretched.

THE SPECIAL SENSES.—The *Argyll Robertson symptom*, i.e., loss of pupil contraction when the eyes are exposed to light after being shut, with preservation of contraction during accommodation, is present in fully 85 per cent. of all cases. It may be present at an early stage. It depends on degeneration of Meynert's fibres, which join the anterior corpus quadrigeminum to that part of the nucleus of the third nerve governing the sphincter pupillæ muscle. The pupils are often very small—*spinal myosis*—owing to the degeneration of the sympathetic fibres in the cord which supply the dilator pupillæ muscle. *Optic atrophy* is present in about 10 per cent. of all cases. It begins early in the disease, and may even be the first symptom. It leads to impairment of vision, which progresses until it ends in total blindness. Ataxia rarely accompanies or follows in cases where optic atrophy is

prominent, but paralytic mental symptoms not infrequently come on. Temporary paralysis of the external muscles of the eye, causing *double vision*, may be present, as may also *ptosis*. *Deafness* may develop, but loss of the sense of smell (*anosmia*) is rare.

Visceral symptoms.—The *bladder* may give trouble at an early stage: there is usually a difficulty in emptying it. Incontinence of urine and cystitis may come on during the late stages. The *bowels* may give similar trouble, but constipation is usually, for a long time at any rate, the only bowel complaint. Loss of control of the rectal sphincter may exist for a time and afterwards disappear. The *sexual desire and power* is decreased or lost, sometimes quite early in the disease. But the most remarkable of the visceral symptoms are the *crises* or severe paroxysms of pain referred to various viscera, of which the stomach stands first in frequency. The gastric crisis consists in intense pains in the stomach, usually accompanied by a gushing vomiting of a highly acid watery bilious fluid. There may be severe hiccough. The attacks are of varying intensity and duration (hours or days), and usually yield only to morphine. The other crises, viz., laryngeal, rectal and urethral, are much rarer.

Vasomotor atrophic symptoms.—There may be local *sweating* or skin *rashes*. A *perforating ulcer*, which usually originates in a corn on the sole of the foot, may be present quite early, but it is rarely seen until late in the disease. It is painless. The teeth may drop out. A perforating buccal ulcer has been described. The *bones*, particularly the long bones, may become brittle and their articular ends absorbed. *Joint affections*, known as *Charcot's joint disease*, are most common in the knees. It may be present early, but it is usually only met with late in the disease. The cartilages and bones are atrophied, and there may be much and rapid effusion into the joint cavity. Suppuration may occur, but pain is absent. Much new bone may be formed and

ankylosis and deformity result. Atrophy of muscles also occurs late in the disease.

The *cerebral symptoms*.—They usually occur late in the disease if at all. Hemiplegia may develop, and there may be melancholia or dementia, but in most cases the brain remains clear and unimpaired until the end.

Diagnosis.—In many cases it is easy to recognise the disease even in its early stage. Lightning pains, the Argyll Robertson pupil, and the loss of the knee jerks is a pathognomonic combination. Other combinations of symptoms, such as ocular palsies and loss of knee jerk, are almost equally so. A history of syphilis is of great value in doubtful cases. *Ataxic paraplegia* is distinguished by the spasticity and the absence of eye symptoms; *cerebellar disease* by the presence of optic neuritis, not atrophy, by the absence of sensory disturbances, by the presence of the knee jerk, and by the gait, which shows a disturbance of equilibrium rather than ataxia.

Prognosis.—Recovery does not occur, but there is always hope of improvement, and an arrest of all, or most of the symptoms may last for years. Optic atrophy is usually accompanied by an arrest of all other symptoms except that mental ones may come on. Complications may arise at any time, and the patient be carried off by pneumonia, tubercle, bladder or kidney mischief, or by sepsis from bed-sores, after he becomes bed-ridden.

Treatment.—A careful study of the etiology of this disease certainly suggests that potassium iodide should be given a most careful and protracted trial in every case. A quiet, well-regulated life should be enjoined, with freedom from any excess—physical, mental or sexual—and the patient should be cautioned against the use of alcohol, even in what he may consider moderate

quantities. A sea voyage is to be recommended where circumstances permit of its being carried out under luxurious conditions.

The *pains*, often so harassing and liable to interfere with the patient's sleep, may be relieved more or less satisfactorily by many different methods of treatment. Amongst the best are rest in bed, blistering, or the application of iodine to the affected region of the back. Galvanism applied along the lines of the painful nerves, and the use of hot baths, are also efficacious remedies. The best way to blister is to apply the button cautery three or four times on each side of the vertebral column. Antipyrin and the other drugs belonging to this group give temporary relief, and the continued administration of small doses of silver nitrate has yielded good results in the hands of several specialists. We have found traction on the legs of the patient beneficial in certain cases, but this method of treatment should be carried out with considerable care, and should not be repeated too frequently. As far as possible, opium should not be given, and it is better to try cocainisation of the cord, although this procedure is not free from risk.

The *crises* demand prompt measures. The *gastric* crises are often very severe. They may be relieved by lavage, although they frequently require the use of opium. *Laryngeal* crises are sometimes benefited by the use of nitroglycerin, and this drug may be tried in any of the other forms of crises present in this disease. A method which obtained considerable notoriety was the hanging of the patient practically by his neck, or obtaining the same effect by bending the patient's back as he lay on a couch. The latter procedure was carried out by the physician seizing the legs, the patient lying on his back, and bending him so as to make the knees approximate as closely as possible to the face. Neither of these methods, by which it was thought that the spinal meninges would be

stretched, and thereby some benefit might result, are free from risk, and they have been given up. The bladder should be carefully watched, retention avoided, and when it threatens, an aseptic catheter should be promptly used. The patient must be warned about the risk of perforating ulcers, and he should be reminded that a corn on the sole of the foot if cut or injured may readily form the starting point of such an ulcer. Frænkel has recommended that the patient should be educated, or, more correctly, re-educated, to perform co ordinated actions, and such exercises have been found of great advantage to the patient in enabling him to get about and to be more independent.

There are few drugs other than potassium iodide which appear to be of genuine benefit in arresting this disease. Many have been tried, and amongst those most frequently recommended are the following:—phosphorus, arsenic, ergot, silver nitrate, calabar bean, and belladonna. The physician should regulate the patient's work, leisure, food and medicine. Many sufferers from the disease have been able to carry on their work for years with little inconvenience.

HERPES ZOSTER—SHINGLES—ZONA.

Etiology.—A poison of unknown origin acting mainly on the ganglia of the posterior roots and upon their processes. It is to be remembered, however, that the sensory nerves are the dendrites of the spinal ganglia and conduct impulses upwards from the skin. It is not therefore so easy to see how an inflammation starting in the ganglia should descend along the sensory nerves to the skin and cause the inflammatory skin lesions characteristic of herpes in the same way as if these nerves were their axis-

cylinder processes. These latter pass inwards into the cord as the posterior nerve-roots.

Morbid Anatomy.—After a period of pain or uneasiness there is a rash, which quickly becomes vesicular. Its distribution is most often in the thoracic region, where its appearance is most characteristic, but it may be in the cervical or lumbar regions or still lower. It has a segmental distribution, one or more adjoining skin fields being affected. It is usually confined to one side of the body. It is more common in children than in adults.

Symptoms.—There may be little irritation or pain, nothing, indeed, beyond the rash, particularly in children, but in adults the pain is often severe, and intractable neuralgia may remain long after the rash has disappeared.

Treatment.—The local treatment consists in guarding the vesicles from injury, and perhaps no method of doing so is so efficacious as the application of collodion painted over a thin film of cotton wool. Where there is much pain, a 10 per cent. or 20 per cent. solution of cocain may be painted on prior to the application of the collodion, or some cocain may even be incorporated in the solution of collodion used. Internally, strychnine is one of the best tonics to administer. Occasional sedatives are necessary for the relief of excessively severe pain.

COMBINED SYSTEM DISEASES.

Some of the diseases already considered show lesions of both the motor and sensory neuronc systems, but the combination is a minor one, since the primary lesions involved remain restricted to either the motor or sensory system during the greater part of the course run by the disease. It

is only towards the latter end that the other system becomes affected, and this extension may be so slight as not to disclose itself by any signs during life. The name combined system disease is therefore not applied to such affections, but to others in which there are signs of involvement of both motor and sensory symptoms from the outset.

ATAXIC PARAPLEGIA.

DEFINITION.—A sclerosis of the posterior and lateral columns of the cord in middle age.

Etiology.—Thirty to forty is the commonest age, and males are far more commonly affected than females. There is a history of exposure to cold, injury, or overstrain, and occasionally to syphilis in some cases, but no closer connection with the disease. The cause is therefore obscure.

Morbid Anatomy.—There is a sclerosis of the posterior columns of the cord, most marked in the dorsal region. Unlike tabes dorsalis there is no involvement of the posterior root zones, and the sclerosis, though similar to, is far less extensive than that seen in Friedreich's ataxia. It is accompanied by a sclerosis in the lateral columns of variable extent and distribution, the pyramidal tracts being frequently, though not always, involved, the direct cerebellar and antero-lateral ascending tract of Gowers being often implicated. There is no meningitis.

Symptoms.—The disease is of slow, insidious onset, some unsteadiness of gait with weakness being usually the first symptom, though the patient may have complained previously for some time of a tired feeling in the legs. Romberg's sign is marked, but there is no pain or other sensory change. The knee jerks are exaggerated and ankle clonus is present. The Babinski sign is generally positive.

Spasticity appears in the legs, but it is rarely so marked as in cases of lateral sclerosis. The inco-ordination and the gait suggest tabes dorsalis but differs from it in showing no jerking movement of the feet or thumping of the heels. The arms may become ataxic and the sphincters usually become affected. Eye symptoms, *e.g.*, the Argyll Robertson pupil, nystagmus, optic atrophy, are rarely seen. As the disease advances, mental symptoms may appear, and the patient becomes bed-ridden.

Diagnosis.—It is distinguished from tabes dorsalis by the difference in gait, the absence of the sensory and the eye symptoms, and by the presence of spasticity; from Friedreich's ataxia by the age of the patient.

Prognosis.—It is steadily progressive until death, which is usually due to some intercurrent affection.

Treatment.—The treatment for spastic paralysis appears to be of most benefit in this disease, and it will be found that residence in a warm climate during the colder months of the year renders the patient much more comfortable. Potassium iodide should not be neglected, and arsenic may be given, more perhaps as a tonic than with any hope that it can exert a specific action. Electricity, at all events galvanism and faradism, should not be used, and strychnine is contra-indicated in all diseases where spasticity is a prominent symptom.

FRIEDREICH'S ATAXIA—HEREDITARY ATAXIA.

DEFINITION—A sclerosis of the posterior and lateral columns commencing at an early age.

Etiology.—It generally begins between the ages of six and fifteen, and very rarely after that of twenty-five.

Males are more frequently affected. Direct transmission from parent to offspring is rare, but it usually affects several members of the same family. It is thus a family rather than a hereditary disease, though a certain weakness in the tracts involved is probably inherited. In some cases the parents have been syphilitic or alcoholic, but there is no clearer relationship between these conditions and the disease.

Morbid Anatomy.—The posterior columns are extensively sclerosed throughout the entire cord, far more so than in ataxic paraplegia. The lateral columns are less involved, generally only an annular sclerosis, which affects the direct cerebellar and Gowers' tracts, and to a less degree the crossed and direct pyramidal tracts. Clarke's vesicular column cells are also involved. Owing to the early age at which the disease begins, and the prominence of the neuroglia overgrowth, it has been suggested that this disease owes its origin to a hereditary defect in the nerve elements and a primary increase in the neuroglia almost amounting to a gliosis of the posterior columns.

Symptoms.—They resemble those of ataxic paraplegia, but show certain differences. The gait is unlike, being swaying and irregular, similar, in fact, to that of a drunken man. The arms, besides being ataxic, show a similar irregularity of movement, choreiform in character. Romberg's symptom is well-marked in some cases and absent in others. The knee jerks are absent. This loss of the deep reflexes is a constant symptom. Nystagmus is present, but the Argyll Robertson pupil and optic atrophy are rare. The speech is usually slow and scanning. Deformities come on sooner or later, talipes being common early and scoliosis later,

Diagnosis.—The differential diagnosis from tabes and ataxic paraplegia has already been given. The drunken gait suggests cerebellar tumor, but the absence of optic neuritis, an almost constant feature of cerebellar tumor, is alone sufficient to distinguish the two diseases.

Treatment.—The treatment may be dismissed in a word. It is impossible to correct the embryonic error probably responsible for this disease, and all that we can do is to obviate deformity by tenotomy or other surgical measure.

CEREBELLAR HEREDITARY ATAXIA.

This disease was first described by Marie. It usually starts after the age of twenty, with typically cerebellar irregular swaying movements in the legs, a "groggy" gait. Some of the symptoms are similar to those of Friedreich's disease, such as nystagmus and the disordered speech, but others are different. Thus the knee jerks are exaggerated and spasticity of the legs appears. Club-foot and scoliosis do not develop.

ANÆMIC AND TOXIC COMBINED SCLEROSIS.

Etiology.—Scattered patches of sclerosis in the white tracts, particularly in the posterior and lateral columns (the grey matter escaping), occur in cases of profound anæmia, whether primary, as in pernicious anæmia, or secondary, as the result of chronic wasting diseases, and in cases of ergotism and pellagra. There have been cases in which the anæmia seemed to bear not a primary but a secondary relationship to the sclerosis, and it is

probable that some toxin is the real determining cause in all cases.

Symptoms.—There may be no symptoms or there may be sensory and motor disturbances (weakness and spasticity) in the legs and later in the arms. The knee jerks are exaggerated. The symptoms will vary with the particular combination of tracts involved. Pernicious anæmia, cancer, diabetes mellitus, and tubercle show a preference for the posterior columns, and ergotism almost repeats the lesions of tabes dorsalis. Exogenous poisons, such as lead, arsenic and silver, affect the cells of the anterior horns, the peripheral nerves, and often also the spinal ganglia. Varying degrees of chromatolysis, atrophy and dislocation of the nucleus are seen. Diphtheria toxin attacks in a similar manner the cells of the anterior horns and the peripheral nerves, the tetanus toxin the anterior horn cells, and the streptococcus toxin the posterior roots and posterior columns.

SCATTERED NON-SYSTEM DISEASES.

There are at least two diseases which may be placed under this heading viz., General Paralysis of the Insane and Disseminated Sclerosis.

GENERAL PARALYSIS OF THE INSANE— PROGRESSIVE GENERAL PARALYSIS— DEMENTIA PARALYTICA.

In its typical form this disease affects the brain primarily and chiefly, though in course of time it extends to the cord and peripheral nerves. A slowly developing atrophy of nerve-cells and nerve-fibres throughout the whole of the nervous system is its leading characteristic, and it would

thus seem to be sharply enough defined from tabes dorsalis. On the other hand, there are many instances in which the diseases appear to be combined. A case may begin as tabes dorsalis and end as a general paralysis, or *vice versa*. In causation (70 to 90 per cent. of each being due to syphilis), pathological lesions, and clinical course, both diseases are closely allied. Mott, Fournier and others hold that etiologically and pathogenetically they are one and the same disease.

Etiology.—Syphilis stands first among imputed causes, there being a history of this infection, either inherited or acquired, in the great majority (70 to 90 per cent.) of cases. Overwork and worry (“burning the candle at both ends”), sexual excess, or blows upon the head, may act as predisposing causes. Males of adult age (thirty-five to fifty) are principally attacked.

Morbid Anatomy.—The dura mater may be thickened and adherent to the cranium, but it is upon its inner surface that it shows the chief change. A pachymeningitis hæmorrhagica interna is often present, principally at the vertex, but sometimes continued over the lateral aspects to the base of the brain. The cerebro-spinal fluid is increased and the pia is thickened throughout the brain and cord, and adherent to the underlying nervous tissue. The brain is smaller than normal, its convolutions being thinner and the sulci wider, particularly in its anterior and middle lobes. In the early stages the white matter of the hemispheres is more atrophied than the grey matter of the convolutions, but the latter becomes more marked as time goes on. There may be little or no change in colour, or the grey matter may be paler. The lateral ventricles are dilated and their ependymal lining has a granular appearance. These characters are even more marked in the fourth ventricle. The nerve-cells of the convolutions and

of the grey matter around the ventricles show various degrees of pigmentation, degeneration and atrophy, and their processes, both grey and white, are shrunken and altered. The surrounding neuroglia cells are enlarged in number and increased in size. There may be scattered areas of softening or necrosis in which much fatty change is present. There is an increase in the number of small blood-vessels and the perivascular lymphatics generally are much dilated and crowded with mononuclear cells. Similar changes are seen in the cord, and degenerative changes in the peripheral nerves. Distinct tract degenerations are frequently present, thus there may be posterior degenerations exactly similar to those of tabes dorsalis, or pyramidal degenerations similar to those of descending spastic paralysis. Ford Robertson describes the presence of a diphtheroid bacillus, to whose action he ascribes the general toxæmia of the disease. There certainly seems to be strong evidence in favour of the opinion that the disease is due to the action of some poison upon the nerve-cells of the central nervous system, leading to degeneration and atrophy in them and in their dendrites and axones, with a consequent hyperplasia of the neuroglial supporting tissue. There certainly is a meningo-encephalitis and myelitis, but the older opinion that the neuroglial change is primary and the ~~nervous~~ secondary has few supporters in the present day.

Symptoms.—The early symptoms present various combinations of motor and mental changes. The former may not be the earliest to appear, but they are the most characteristic. Tremor of the lips and tongue in speaking or in showing the teeth are noticed. The tremor may extend to the face muscles. The speech is slow and hesitating, with a tendency to drop syllables. Labials, linguals and dentals give most difficulty in pronunciation. Certain words and phrases are particularly difficult, e.g.,

"British Constitution," "Artillery," "Round the rugged rocks the ragged rascal ran." Eye symptoms, too, may appear. There may be temporary or permanent paralysis of one or other of the ocular muscles, inequality of the pupils, the Argyll Robertson phenomenon, or in a small number of cases optic atrophy. Some of these motor symptoms may be present for long periods, even years, before mental symptoms appear, or they may not come on till afterwards. The mental symptoms are very variable, but in all cases they amount to a disturbance of the mental balance. The patient may show fits of excitability or irritability or unusual depression. Periods of apathy and great restlessness may follow one another without any apparent cause. He often shows a loss of the sense of proportion he previously possessed, and gives things fictitious values. He may launch out into wild speculation or commit pecuniary or moral extravagances. This state of mental exaltation which fosters ideas of grandeur is not so pronounced in the early stages as it gradually becomes later on, when he suffers from great restlessness, sleeplessness and noisy excitement. This condition of "*expansive delirium*" may be slight, may disappear, or may never appear at all, and there may be instead a state of mental depression amounting to melancholia or hypochondriasis. Speech difficulties gradually become more marked. Writing is difficult because of the unsteadiness of the hands and confusion of ideas; words or syllables may be omitted. The knee jerks may be exaggerated, absent or different on the two sides. Convulsive fits may come on, and recurrent attacks of aphasia sometimes appear. Bladder and rectal troubles gradually arise. In the last stages the patient becomes helpless, bed-ridden, callous and indifferent to everything—a mere senseless, helpless log, living, it is true, but no longer human. Bed-sores are liable to appear. Death occurs

from exhaustion or from pulmonary or other intercurrent affection.

Treatment.—There is no disease so hopeless as regards treatment, or so dire in its final results, as general paralysis. The anticipation that serum therapy may yield treatment of real benefit appears to be supported by the recent work of Dr Ford Robertson and his collaborateurs. The diphtheroid organisms separated post-mortem from the central nervous system of general paralytics and injected into sheep have yielded a serum which in a proportion of cases has given encouraging results, although at present we cannot write more definitely of its success. Potassium iodide, it is hardly necessary to say, is worth a trial, but the only drugs to which we need refer are hyoscin and other sedatives, including bromides, which are of special value where there is much excitement. The latter drug appears to have a very definite beneficial effect in the epileptiform seizures so commonly found in general paralysis. The patient should be carefully dieted, his life well regulated, and all worry or mental strain absolutely prohibited.

MULTIPLE CEREBRO-SPINAL SCLEROSIS— DISSEMINATED SCLEROSIS—INSULAR SCLEROSIS—SCLEROSE EN PLAGUES.

DEFINITION—A chronic disease of the brain and cord in which scattered patches of sclerosis replace more or less completely the nerve elements.

Etiology.—Its cause is obscure. It usually comes on after puberty and sometimes in middle life. It has followed more or less closely in the wake of certain of the infectious diseases, particularly scarlet fever, of chills and injuries. Syphilis is held by some to have a causal influence,

and by others a defective embryonal development based upon a hereditary or family history is blamed.

Morbid Anatomy.—The patches vary greatly in size, from minute microscopic foci to large areas of about one to three inches in diameter. Their shape is irregular. Their consistence is distinctly firmer than that of the normal nervous tissue. Their colour is grey, slate-coloured or greyish-red. It is their distribution, however, which is their most striking feature. They may predominate in the brain or in the cord, but in most cases they are irregularly distributed throughout the entire central nervous system, involving both the white and grey matter, but particularly the former. They may be superficial or deeply seated. In the brain cortex they are often small, while in the tissues around the ventricles, in the basal ganglia, pons and medulla, they are often large. In the cord the cervical and lumbar enlargements seem to be the favourite seats, but they may be situated at any level, from the medulla to the cauda equina. At one level they may occur in the posterior columns, at another in the anterior tracts, and at another in some or all of the cord tracts combined. Their distribution has no relation to the nerve tracts or the individual nerve structures, and it has therefore been suggested that it may be related to the blood-vessels, and some recent writers support this opinion. Microscopically there is an increase in the neuroglia and connective tissue and a loss in the myelin sheaths of the nerves, so that in many parts of the patches axis-cylinders, entirely destitute of myelin sheaths, are seen running through the sclerosed tissue. It is undecided which of these two changes, viz., the degeneration of the myelin sheath or the sclerosis, is the primary one. It is remarkable that these naked axis-cylinders may persist for long periods and discharge their functions more or less perfectly, though they ultimately disappear, hence secondary degenerations are slight.

Symptoms.—Chârcot divides the disease into three stages which may be long protracted. The first stage begins with stiffness and weakness in the legs and a marked increase in the reflexes, and proceeds to the disablement of the patient through the rigidity of his muscles. The second stage continues the spastic rigidity of the muscles and terminates with the loss of the organic reflexes. The third stage continues from the loss of the organic reflexes till death from bed-sores or other intercurrent mischief ensues. The individual symptoms vary greatly according to the site of the sclerotic patches, but among the most important of them are the following:—(1) *Intention tremor*.—Quick, jerking movements occur in the arms or in one arm when the patient tries to perform a muscular action. They may appear also in the legs and head. They are increased by nervousness and are independent of his will; the more he tries to control them the worse they become. The usual test is to give the patient a glass of water to drink. The jerking movements increase as he lifts the glass to his lips, and often cause him to spill a lot of the water, or even prevent him bringing the glass to his mouth. If he succeeds, the movements quiet down when he begins to drink. The inco-ordination is not usually so marked in the legs. When the patient is at rest in the recumbent position and during sleep the movements entirely cease. (2) The *speech* becomes *scanning*, slow and syllabic, or the ends of the words may be cut short. Memory fails, mental dulness, and even insanity, may supervene. (3) *Nystagmus*, lateral rarely vertical, is almost always present in both eyes. It is a rapid oscillation of the eyeballs to one or other side. Other eye symptoms may be present, *e.g.*, optic atrophy, paralysis of eye muscles if a patch involves the chiasma or the nuclei of the optic or of any of the eye muscles. (4) Sensory symptoms are often absent, but headache and vertigo are often severe. (5) The control of the sphincters is lost in the last stage of the disease.

Diagnosis.—When volitional tremor, nystagmus and scanning speech are present the diagnosis is easy. Hysteria is distinguished by the simulated tremor being not volitional, and by the absence of nystagmus and other signs.

Prognosis.—The disease is generally progressive and proves fatal in five to six years, but it may be arrested for a considerable time in favourable cases.

Treatment.—Many of the milder cases, which the older physicians would not have designated instances of disease at all, are amenable to a course of treatment which may be called the “rest cure.” Sometimes potassium iodide yields hopeful results, and it has been found that Swedish exercises are beneficial. Arsenic and silver have been recommended, although it is impossible to speak of either of those remedies with great enthusiasm.

TUMORS OF THE BRAIN AND CORD.

A. THE CORD.

Tumors in the cord are rare. They are similar to those of the brain. The symptoms to which they give rise depend upon their situation and size. They are similar to those of hemisection or of complete transverse section of the cord. When the tumor reaches the surface it causes a localised meningitis and pressure upon spinal nerve-roots. Compare with tumors of the spinal membranes, *vide* pages 848 to 856.

B. THE BRAIN—INTRACRANIAL TUMORS.

Chronic tubercle, syphilitic gummata, and cysts form masses which give rise to symptoms similar to those of true tumor, and hence they are included here. *Sarcoma*

is the commonest of the true tumors. It is often primary, arising in the membranes most frequently, and may reach a large size (a hen's egg or bigger) without giving rise to secondary growths, and without causing any further trouble than its pressure inflicts. Such tumors have been frequently removed with success. A more grave form arises in the brain substance, constituting a large rapidly-growing and infiltrating tumor. *Glioma* originates always in the brain substance and forms a varying-sized, firm or soft ill-defined tumor. It may grow very slowly and is apt to have hæmorrhages occurring within its substance. *Cancer* is rarely primary, but not infrequently secondary to a tumor elsewhere. *Tubercle* forms a single or multiple growth, varying in size from a pea to that of an orange. The centre is caseous and the margin grey and well-defined. The cerebellum is its commonest site. It is probably the commonest of all the intra-cranial growths. It occurs in the young, generally under ten and rarely over twenty years of age. The *gumma* usually grows from the pia mater and causes pressure of the brain substance. It rarely reaches a large size. It may be single or multiple. It often implicates the region of the pons, hence ptosis and strabismus are common. The *cysts* may be parasitic, *e.g.*, hydatid, or the result of hæmorrhage or of softening.

Symptoms of Intra-cranial tumors.—There are both general and focal symptoms.

GENERAL SYMPTOMS.—(1) *Headache* is frequent. It may be dull and continuous or sharp, piercing and paroxysmal. It may be felt all over the head, or limited to some part, or even to the course of an individual nerve. (2) *Vomiting* of a cerebral type, *i.e.*, not related to meals, is an important and frequent feature. It may be very intractable, particularly in tumors of the cerebellum and pons. (3) *Optic neuritis* occurs in four-fifths of all cases, being constant in tumors of the corpora quadrigemina, almost so in

those of the cerebellum, and absent in nearly two-thirds of those of the pons, medulla and corpus callosum. It is most frequent when the tumor is a glioma, sarcoma, gumma, or cyst, least so when it is a tubercle. It is thought to be due to increased intra-cranial pressure from dilatation of the lateral ventricles caused by pressure on the veins of Galen. These three symptoms are often spoken of as the tripod of cerebral tumor. (4) *Giddiness* is often an early symptom. It varies in degree and may be present only, or most marked, when the patient rises suddenly from the recumbent posture, as in getting out of bed, or turns his head quickly. (5) *Convulsions*, either general or Jacksonian, may be present. (6) *Pulse and respiration* may be slow. (7) *Mental disturbances*, e.g., dulness, heaviness, slowness of speech, loss of memory and emotional tendencies may occur.

FOCAL SYMPTOMS.—The cranium should always be carefully percussed, and any marked local tenderness is in favour of a superficially placed tumor in that situation. This is a useful general guide, but definite disturbance of function referable to certain areas presiding over such functions is of far more importance. Tumors, like other lesions, e.g., foci of softening or hæmorrhage, particularly when situated in the cortex, cause in their earlier stages irritative changes, and in their later stages paralytic symptoms, from the nature of which the site of the lesion may be often diagnosed with a fair amount of certainty. The irritation causes spasm in a group of muscles it may be, e.g., if the spasm affects the muscles of the face, angle of the mouth or tongue, the lesion is probably in the lower third of the ascending frontal convolution; if it affects the hand, wrist or arm, it is in the middle third; if it affects the toes, ankles or legs, it is in the upper third. It may extend to other areas after affecting any of these in the first place. Sequin gave the name of *signal symptom* to this condition, i.e., to a spasm beginning in a certain muscle group and

extending to others, and this group of clinical symptoms constitutes the Jacksonian form of epilepsy. This signal symptom generally points to the part first affected, *i.e.*, to the site of the tumor or other lesion. The earlier symptoms are *irritative*, *i.e.*, there is spasm or convulsive movements of the group of muscles affected, and the later symptoms are *paralytic*. The lesion may be in the cerebral cortex, the internal capsule or basal ganglia, in the crura, in the corpora quadrigemina, in the pons, in the medulla, or in the cerebellum, and the particular function involved will enable the observer to say, in many cases at any rate, in which of these positions it is. Thus if the lesion is in the *prefrontal region* there may be no definite symptoms; but there is, on the other hand, likely to be some disturbance in the mental state of the patient. The mental characteristics may be altered, particularly if the left side is involved. Torpor and sluggishness of thought are common. If the *inferior left frontal convolution* (Broca's) is involved, motor aphasia is present; if the *ascending frontal convolution* is implicated the results will vary with the part of it affected. If the *parieto-occipital lobe* is involved there may be no symptoms, if the *angular gyrus* on the left side is affected there is word or mind blindness. If the *occipital lobe* is implicated there may be hemianopsia and also word or mind blindness. In the *temporal lobe* there is often no result, but if the lesion involves the auditory sense area (the first and transverse temporal gyri on the left side) there may be word deafness. Tumors confined to either the *corpus striatum*, *lenticular nucleus*, or *optic thalamus*, if small, may produce no results, but whenever they extend to the *internal capsule* they are apt to produce hemiplegia if they involve its anterior two-thirds, or hemianopsia and hemianæsthesia with optic neuritis if they injure the posterior third. Destructive lesions of the internal capsule cannot be distinguished from similar lesions of the Rolandic cortex, but irritative lesions may be. If situated in the cortex

the resulting convulsions are tonic and then clonic; but if situated in the centrum ovale or internal capsule they are tonic only. When the lesion destroys the *retro-lenticular part* of the *capsule* there is hemianæsthesia, hemianopsia, disturbances of hearing, and, if it extends into the optic thalamus, athetosis or chorea. Lesions limited to the *crura cerebri* are rare. They often affect both upper and lower motor segments, producing an alternate or crossed paralysis, e.g., a hemiplegia of the opposite side of the body from the lesion and a paralysis of the third nerve on the same side. Hemianopsia in the opposite halves of the visual fields will be present if the optic tract which crosses the crus is involved. Anæsthesia will also be present if the *tegmentum* be implicated. An oculo-motor paralysis of one side and a hemiataxia of the opposite side are said to be characteristic of a tegmental lesion. Lesions limited to the *corpora quadrigemina* are rare, but lesions in their neighbourhood may involve either the superior or the inferior bodies. In the former case some of the eye muscles are weakened, the pupils being dilated and not reacting to either light or accommodation; in the latter case, hearing may be defective on the side opposite to the lesion. Lesions of the *pons* and *medulla* are characterised by profound coma and general muscular paralysis with a tendency to tonic or clonic spasms. Pin-point pupils are generally seen, and also a high temperature in the case of a lesion of rapid onset, such as a hæmorrhage. Small lesions may cause hemiplegia, and it is important to remember that the nerve-fibres from the face centre decussate in the *middle of the pons* on their way to the seventh nucleus, hence a unilateral lesion above this, *i.e.*, in the upper half of the pons, causes paralysis of face, arm and leg on the opposite side, whilst one below this, in the lower half of the pons, causes paralysis of the face on the same side as the lesion, and of the arm and leg on the opposite side. The patient often has a tendency to fall towards the

side on which the lesion is situated, whilst simple hemi-ataxia is still more common. The symptoms resulting from implication of the different cerebral nerves are given under these nerves. When the tumor or other lesion, *e.g.*, abscess, hæmorrhage, involves the *cerebellum*, there may be instability when attempting to stand or walk, although when lying down delicately co-ordinated movements may be performed. The gait is like that of a drunken man. The inco-ordination is of a coarser kind than in lesions of the cerebral cortex, and it has been suggested that it may be caused by the withdrawal of the influence of the cerebellum upon the cerebrum. Paresis or paralysis, which may occur, is thought to be due to pressure upon the pons or medulla. Convulsions may occur on the same side as the lesion. There may be tonic rigidity of the neck muscles. The knee jerks are apt to be increased on the same side, though they may be unaffected. The pupils vary. Nystagmus is common. Glycosuria may be present. Sudden death may occur. If the lesion be situated in one hemisphere and does not involve the vermis, there may be no symptoms, for other parts of the brain appear to be able to take on the functions of parts of the cerebellum.

Treatment.—The treatment for every case of cerebral tumor should, in the first place, be antispecific, because in those which are syphilitic the most pronounced benefit follows long continued treatment along these lines. Headache is relieved by the local use of the icebag, and by sedatives, especially the bromides. In a very severe case the only sedative which renders the patient's life endurable is opium, and there is no reason why it should be withheld. A certain number of cases of cerebral tumor are operable, and, fortunately, whether we are able to diagnose the exact position of the tumor or not, trephining the skull will be found of the greatest value in preventing the advance of optic

neuritis which is so commonly present in cases of intra cranial tumor. Whether it be that optic neuritis implies greatly increased pressure of cerebrospinal fluid, or whether in a certain number of cases, at all events, there is a toxin responsible for the change in the optic papilla, the benefit following trephining as regards the patient's sight is very pronounced, and fully warrants the risk of the operation. It will be found also, that the cerebral vomiting and the headache are frequently relieved after some cerebro-spinal fluid has been allowed to escape. Lumbar puncture is certainly worthy of trial in cases where, from want of localising phenomena, it is difficult for the surgeon to decide where to trephine, but the removal of fluid by lumbar puncture, although a much safer operation, and one which can be repeated as often as desired with little inconvenience to the sufferer, has not afforded the same degree of benefit as the major operation.

DISORDERS OF SPEECH.

INTRODUCTION.—The word *aphasia*, though originally more restricted, is often used to include all forms of speech disorder.

Etiology.—Destruction of certain brain centres, or of the nerve paths which connect them, produces disturbances of speech, which vary according to the position of the lesion. This latter may be a tumor, a softening, a hæmorrhage, an abscess, or a degeneration caused by a toxic agent, such as alcohol, morphia, cocaine.

Morbid Anatomy and Symptoms.—Morbid changes characteristic of destructive lesions of the brain, e.g.,

softenings, hæmorrhages, tumors, occur in the definite areas which govern speech. Spoken words reach the temporal lobes from the ears, those from each ear going to both sides of the brain. From these primary auditory centres impulses are sent to the *hearing* or *auditory speech centre*, which is situated in the first or superior temporo-sphenoidal convolution on the left side of the brain, where they are interpreted into the ideas associated with the words.

From this auditory speech centre, which constitutes the memory for words heard, impulses pass along fibres which run in the white matter of the island of Reil to the posterior part of the left third or inferior frontal convolution or Broca's convolution, which is called the *motor speech centre*, and excite the motor speech memories stored therein. Bastian calls it the glosso-kinæsthetic area. Motor impulses are sent from this centre by means of the pyramidal fibres to the nuclei of the cranial nerves supplying the muscles which are used in the production of articulate speech, viz., those of the larynx, pharynx, palate, tongue, lips, jaws and respiration. There may be other paths which can be used when the left pyramidal fibres are interrupted. There is reason to believe that Broca's convolution is connected by commissural fibres through the corpus callosum with the corresponding area of the right frontal lobe and that messages unable to pass directly may cross over to the opposite side and then be transmitted through the right pyramidal tract to the nuclei of the same cranial nerves. In like manner, written words pass to the visuo-sensory centres in the posterior parts of both occipital lobes, thence to the *visual word or speech centre* in the angular and supra-marginal convolutions on the left side. By this centre words or symbols seen are interpreted into ideas and stored up in the memory. The seat of the intellect is not yet known, but many think that it is constituted by a combination of the three speech centres—the auditory, visual and

motor. In expressing thoughts in words, the memories of the sounds it is necessary to use are revived by the auditory speech centre, whence impulses pass to the motor speech centre and thence to the nerve centres, nerves and muscles used in the production of articulate speech. In reading aloud the impressions of the words enter by the eyes and reach the visuo-sensory centres, then the visual word centre, whence they travel across to the auditory word centres and are transmitted as before to Broca's convolution and to the muscles. Some think that Broca's convolution controls the movements necessary not only for speaking words but also for writing them. On the other hand, the majority hold that there is a special writing centre situated in the posterior portion of the second or mid-frontal convolution. In writing from dictation the impulses pass from the ears to the auditory word centre, then to the visual word centre, where the shapes of the letters composing the words are revived. Impulses carry them by association fibres to the writing centre, which controls the movements involved in writing just as Broca's does those of speech. The speech mechanism may be said to be complete after the development of the associations which underlie reading and writing. The cortical speech centres, viz., auditory, visual, motor and writing, occupy parts of the brain bounding the Sylvian fissure and all receive their blood supply from the Sylvian artery. They may all be involved by lesions of that artery, or the motor only, lying in front of the fissure, or the sensory only, lying behind the fissure.

Motor aphasia.—It is rare for a lesion to affect the writing centre only, causing loss of power of writing, or *agraphia*. The motor speech centre (Broca's convolution), on the other hand, is frequently affected. This constitutes *motor aphasia*. It is usually, though by no means always, accompanied by *agraphia*, which is itself a form of motor aphasia. In this cortical motor aphasia the patient is unable to speak spontaneously or to repeat

words, though he may be able to pronounce perfectly one or two simple words or phrases, like "yes" or "no," which is thought to be due to the activity of the corresponding region of the right side. His mind is clear, and he understands what is said to him. He knows the right word by which to name an object, but cannot himself pronounce it. His power of reading is partially or entirely unimpaired. When the patient can pronounce words quite well, using not the words he wishes but inappropriate and senseless ones, the condition is called *paraphasia*. A subcortical motor aphasia also occurs. When the lesion is in the white matter of the left side, close to Broca's convolution, the loss of power of speech may be as complete as before but there is no *agraphia* if the hand is not paralysed, and the mental processes are undisturbed. A lesion lower down, *e.g.*, near the knee of the internal capsule on either side, causes slight disturbances of speech due to the weakness of the lip and tongue muscles of the opposite side. This often amounts to nothing more than the blurring of the speech noticed in most cases of apoplexy. Improvement frequently follows in a short time, probably due to Broca's convolution sending its impulses over to the right side as already explained. When the lesion in the internal capsule is bilateral, there is interference with the muscles of articulation on both sides (*pseudo-bulbar paralysis*). Bastian gave the name *aphemia* to a difficulty or inability to utter or articulate words. Disturbances of speech may also arise from lesions of the lower motor neurones, *viz.*, the nuclei of the motor nerves in the pons and medulla, and the peripheral nerves arising from them and going to the muscles used in the production of speech. Such lesions are often bilateral, as in bulbar paralysis. They may cause speechlessness (*anarthria*) or merely disturbances of articulation, the patient using the correct words and arranging

them rightly in order, but being unable to pronounce them properly (*dysarthria*).

Sensory aphasia.—In sensory or amnesia aphasia or amnesia, either the auditory or visual word centres or the tracts which connect them are diseased.

The auditory word centre.—Complete destruction of the first temporo-sphenoidal convolution causes inability to understand spoken words, *i.e.*, word deafness. The patient can voluntarily pronounce words, but they convey no meaning to him. He cannot repeat words nor write to dictation. He may be able to copy. He soon becomes speechless. Partial destruction of the centre causes partial loss of memory for words. He may be able to understand what he hears and reads, to repeat words and to write to dictation, but cannot speak well because of the loss or transposition of certain words. Bastian calls this condition “*amnesia verbalis*.” When the lesion involves not the centre itself, but the afferent paths leading from the two primary auditory centres to it, *i.e.*, in the white matter beneath it, the patient is unable to understand what he hears. He cannot repeat words or write to dictation, but he can copy, write and speak voluntarily, and read perfectly.

The visual word centre.—Destruction of the centre in the angular and supra-marginal convolutions causes inability to read anything written or printed, *i.e.*, *word blindness*. He cannot write or copy, but he can understand what he hears, and can speak voluntarily. If the afferent tract going to the visual word centre be involved he cannot understand written or printed words, but he can hear, speak, and write, though he cannot understand his own writing. It is rare to find one of these centres alone involved, whereas combinations are common. Blindness and word deafness are often combined, and they may be associated also with inability to recognise the nature of objects seen, *i.e.*, *mind blind-*

ness, and with inability to understand not only words but other sounds heard; *i.e.*, *mind deafness*. The term *apraxia* is used to designate a condition in which the patient is unable to recognise not only an object but also its uses and relations. It may occur alone, but is usually associated with forms of aphasia.

Prognosis.—In children the outlook is good, since the opposite hemisphere can readily be educated. In adults it is less hopeful. On the whole it is better in sensory than in motor aphasia. Important questions may arise in medico-legal cases, particularly as to the validity of wills executed by aphasic persons. Such a document will probably be held to be valid if the patient retains sufficient mental capacity to appreciate the details of the procedure.

GENERAL OR FUNCTIONAL DISEASES.

A GROUP OF DISEASES IN WHICH THERE IS NO
CONSTANT OR WELL MARKED STRUCTURAL
LESION.

I. PARALYSIS AGITANS—PARKINSON'S DISEASE —SHAKING PALSY.

DEFINITION.—A chronic disease of the nervous system characterised by tremors, muscular weakness and rigidity and a peculiar attitude.

Etiology.—It rarely occurs under forty and is more common in men. There is often a family history of other nervous affections. Exposure to cold, wet, fatigue, fright, injury, business worry and fevers have all been regarded as causes.

Morbid Anatomy.—Patches of sclerosis in the medulla and cord and degenerations of the ganglion nerve-cells of the cerebral cortex have been described in some cases, but there is no constant lesion. The changes are probably those of senility coming on prematurely and more pronouncedly than usual.

Symptoms.—A gradual onset is the rule, an abrupt one the rare exception. The four prominent symptoms, not always present together, are tremor, weakness, rigidity and attitude.

Tremor.—This usually begins in one hand, to which it remains limited for a long time. It consists in movements of the thumb and forefinger as if the patient were rolling a pill, or in pronation and supination of the hand at the wrists. In the legs the movements are best seen at the ankle-joint. Shaking of the head is less frequent and is generally vertical, not rotatory as in senile palsy. The movements occur at the rate of about five per second. They are involuntary, but can be controlled by the will, at least for a time, since they may cease for a little when he makes a voluntary movement, only to return, however, with increased intensity. They are exaggerated by emotion, lessened in the attitude of repose, and cease entirely during sleep. The handwriting is at first good but soon becomes tremulous. The movements generally become more marked as time goes on. In some cases they are absent altogether.

Muscular weakness and rigidity.—There is always some weakness: it may precede the tremor, but generally comes on and advances along with it. The muscular movements are slow and stiff. The joints become flexed and lead to the peculiar attitude.

The attitude and gait.—The head is bent forward, the back is somewhat bent, the elbows flexed and the face immobile, expressionless and mask-like (the so-called

Parkinson's mask). The intellect is clear and in marked contrast with the vacuous expression. He speaks slowly in commencing a sentence and finishes it quickly as if in a hurry, and the voice has often a shrill pitch. He rises from his chair in a stooping posture, slowly and with difficulty. In beginning to walk his steps are slow, but they soon become short and quick, which Trousseau described as running after his centre of gravity. This is termed propulsion or the festinant gait. If he be pulled backward (retropulsion) his steps also increase in rapidity until he falls. The reflexes and electrical reactions are normal. The patient usually complains of cold, and sometimes also of heat, either generally or locally. Sweating may be noticed. In the later stages the flexed joints may become ankylosed.

Diagnosis.—There is no difficulty in typical cases. The attitude and mask-like expression are not given in any other disease. Senile tremor is less marked in the hands and does not show the pronator and supinator movements, while it is more marked in the head. In disseminated sclerosis, the movements are jerky, and there is nystagmus, scanning speech, or exaggerated reflexes. Tobacco and alcoholic tremor occur chiefly on movement.

Prognosis.—The disease lasts for years, and while progression is generally steadily downwards, periods of improvement may occur.

Treatment.—Attention to the general health, the use of sedative remedies, which sometimes control or at least alleviate the shaking movements, and the practising of rhythmic exercises are the indications for treatment. Hyoscine and hyoscyamine have proved of benefit in some cases, and arsenic is a useful tonic, but no drug as yet in use has any specific action in the disease.

II. ACUTE CHOREA—SYDENHAM'S CHOREA— ST VITUS' DANCE.

DEFINITION—A disease commonest in children, characterised by involuntary jerky movements of the muscles of the face or limbs, closely related to acute endocarditis and rheumatism.

Etiology.—*Age and Sex.*—It is most common between the ages of five and fifteen, and much more frequent in girls than boys, particularly after puberty. Highly strung, nervous children are particularly liable to the disease, in whom it may follow a fright, a scolding, an injury, overwork at school (school-made chorea), or occasionally by imitation of another child. *Pregnancy.*—It is not uncommon during pregnancy, particularly in the early months, and may become so acute as to endanger life. *Rheumatism.*—The general connection with rheumatism is close. There may be a history of rheumatism in the family, a previous record of it in the patient, or rheumatism and chorea may exist together, and it may be difficult to say which of these mischiefs began first. *Endocarditis.*—This is the most frequent of all the associated conditions, and some writers even regard endocarditis as the cause of acute chorea. It is thought that pieces of the cardiac vegetations break off and pass as emboli into the cerebral vessels, which may thus become blocked in places, as a result of which the nerve-cell degenerations arise and cause the choreic symptoms.

Morbid Anatomy.—There is no constant lesion, though some form of morbid change capable of causing diminished nutrition in the areas supplied by the affected vessels has been found in many cases. Among such changes the following may be mentioned:—embolic or thrombotic processes, minute hæmorrhages, and leucocytic infiltration or hyaline degeneration of the vascular walls. A choreic condition has been produced experimentally in

animals by the injection into the blood-stream of fine particles of sand (Rosenthal and Money) or of the diplococcus rheumaticus of Poynton and Payne, and in many cases there is, as already mentioned, a coincident endocarditis or arthritis. But in other cases no vascular lesion or lesion of any kind has been discovered, and hence the disease is still most generally regarded as a functional one due to some instability in the motor nerve mechanism. This instability may exist in the motor cortex, in the basal ganglia, in the anterior horn cells of the cord, or in the pyramidal fibres.

Symptoms.—Jerky, irregular, inco-ordinate movements appear, usually first in the hands and arms, then in the face, and later in the legs. The movements may show all degrees of severity from merely slight twitchings about the eyes or mouth to violent muscular spasms of the body sufficient to tumble the patient out of bed. They are involuntary and are increased by nervousness and voluntary effort, but usually cease during sleep. They are generally unilateral (hemi-chorea), and when bilateral are often greater on one side than on the other. Mild degrees cause restlessness ("fidgets") or awkwardness of movement, articles may be knocked over, and if the patient be asked to put out the tongue, it is suddenly jerked out and as suddenly withdrawn, or it is bitten by a sudden closing of the jaws. The same irregular, jerky clonic spasms are seen in the hands and face, and the patient exhibits muscular movements in excess always of those which are necessary for the proper performance of any particular act. The speech, respiration and deglutition are unaffected. In more severe forms the movements are so extensive that they interfere with walking or with the performance of any steady purposive actions. Speech is affected, causing either a hesitancy, an incoherency, or inability to speak at all. Respiration may assume a panting character at times, and there may even be difficulty

in swallowing. Muscular weakness appears after a time, slight at first but increasing afterwards, until it may amount to distinct paresis. In the most severe forms of the maniacal chorea the movements exhaust the patient, prevent sleep and gravely threaten life. They occur most frequently in adult women, particularly during pregnancy, and may develop out of the ordinary form. Sensory symptoms are generally absent, though pain and tenderness in the affected limbs are sometimes complained of. Fever is mostly absent except in cases of maniacal chorea. Psychical disturbances are frequent. The temperament may change, the child, previously quiet and docile, becoming peevish and irritable, inattentive and forgetful. In severe cases maniacal delirium may come on.

Diagnosis.—Many cases are easily told at a glance. In others the choreic movements are brought out by asking the patient to perform some definitely co-ordinated action such as putting out the tongue, drawing a circle with either the fingers or the toes, tying knots on a string. In all these actions the patient expends an excess of muscular energy, the movements are jerking and in excess of those required to perform the specified acts. Great care must be exercised in mania coming on in pregnancy, as cases of a choreic character may be mistaken for true mania.

Prognosis.—Most cases recover in from eight to ten weeks. Exceptional ones last longer, months or even years, but ultimately recover. A few, particularly those in which the movements are very violent or those in which cardiac lesions are present, may take a downward and fatal course.

Treatment.—The child should be kept at rest in bed and all mental and physical efforts stopped. Where there is

much excitement, isolating the patient by placing screens round the bed (if in a hospital ward) yields excellent results. Any peripheral cause of irritation should be removed and the patient should be fed on a simple but sufficient diet. In cases of an endocarditic nature salicylates should be administered.

There are various remedies which have been found of use in chorea, and especially arsenic, which may be given in gradually increasing doses until the movements subside, but care must be taken not to run any risk of arsenical poisoning. Antipyrine and bromides are often prescribed in combination, and many sedatives such as belladonna and chloral are worth a trial where the first mentioned drugs fail to benefit. The importance of the causal factors—overwork and excitement in school life—should not be forgotten when dealing with highly strung children, and a child who has once had chorea should not be subjected to the risk of a relapse by any want of care and attention in regard to lessons. In maniacal cases and where the movements are very violent the patient should sleep on a mattress on the floor, and should be prevented from bruising the limbs by a judicious arrangement of pillows. Sometimes chloroform anæsthesia is necessary so as to give the sufferer a period of restful sleep, and a nutritious diet must be given to support strength. Wrapping up the patient in a wet sheet similar to the treatment adopted for typhoid fever has been recommended for soothing great excitement. In chorea gravidarum, where the movements are often very violent, labour may require to be induced.

III. CONDITIONS ALLIED TO CHOREA.

CHRONIC CHOREA—HUNTINGDON'S CHOREA.

This disease is markedly distinguished from acute chorea, with which it has no connection, by the late

period of its onset, viz., at or after middle life, by its strong hereditary character in many cases (sometimes attacking four or five generations), and by its psychical disturbances. The speech is slow and slurring and there is a gradual and progressive diminution of the mental powers towards dementia. The movements are irregular and inco-ordinate rather than choreic. They appear in the hands and arms before the legs. When they affect the latter they cause an unsteady, swaying gait like that caused by alcoholic intoxication.

SENILE CHOREA.

Choreic movements sometimes appear in middle-aged or old people without any mental impairment. There is no hereditary tendency, and the cases remain mild.

RHYTHMIC OR HYSTERICAL CHOREA.

This causes rhythmic movements in any group of muscles, *e.g.*, the abdominal muscles producing the salaam movements, or the head and neck muscles, causing rhythmic nodding, &c., of the head.

CHOREA MAJOR.

This is the original St Vitus' dance, so named because of the dancing, jumping and gesticulating movements indulged in by the pilgrims in the Middle Ages to the chapel of St Vitus in the Rhine provinces during periods of religious excitement. It is a form of hysteria.

SALTATORY SPASM.

This is another form of hysterical movement, apt to run in families, in which the patients jump or spring violently up whenever they attempt to stand, and sometimes also whenever they are excited. There is a tend-

ency to utter loud cries or a frequent repetition of a single word (echolalia). It may be transitory or last for years.

HABIT SPASM.

This consists in quiet spasmodic movements of the head, face or body muscles, *e.g.*, winking, twitchings of the lips and other facial grimaces, shaking of the head, shrugging of one shoulder, &c. They are common in nervous children from seven to fourteen years of age, and affect girls oftener than boys. They are exaggerated during times of fatigue or excitement. They are usually transient, but it is not uncommon for some twitching, particularly of the eyelids, to persist permanently.

IMPULSIVE TIC (GILLES DE LA TOURETTE'S DISEASE).

This is a hysterical affection of graver character in which, in addition to more violent muscular spasms, there are peculiar psychological disturbances. The patient gives forth explosive utterances like a bark, inarticulate cry or loud shout, or mimics a word over and over again (echolalia), or utters oaths or obscene words (coprolalia). In addition, there is a tendency to delusions which cause the patient to touch certain objects or count a number of times before performing any action however trivial.

IV. EPILEPSY.

DEFINITION—A sudden loss of consciousness with or without convulsions.

Etiology.—*Predisposing Causes.*—(1) *Age.*—Most cases begin in youth, more than three-fourths of them appearing in patients under twenty years of age. (2) *Sex.*—It affects

the sexes nearly in equal numbers. (3) *Heredity*.—It may not itself run in families, but it tends to attack those with a family history of marked neurosis, particularly hysteria and insanity. (4) *Chronic alcoholism* in the parents appears to favour epilepsy in the offspring. (5) *Syphilis* is believed to have a similar influence. *Exciting causes*.—Fright, fevers, injury, reflex irritation, e.g., a tight prepuce, worms, dentition, errors of refraction, a foreign body in the ear or nose, toxic agents such as alcohol, lead and uræmia, have all been blamed as direct exciting causes of epilepsy, and any one of them may be followed by epileptiform seizures, but whether one or all of them can produce true epilepsy is not really proven. True epilepsy may appear in old people without obvious cause. Organic lesions of the brain and its membranes generally cause the Jacksonian type of epilepsy.

Morbid Anatomy.—No definite lesion has yet been found. Vasomotor spasm was thought for a time to be the probable cause of the convulsive fits, but this idea has been given up of late. Degenerative changes have been described in the cortical and other nerve-cells, but their influence upon the causation of the disease is unknown.

Symptoms.—I. THE GRAND MAL OR MAJOR EPILEPSY.—The symptoms of this form of epilepsy are conveniently grouped under three heads in relation to the fit, viz., before, during and after it. (1) *Before the fit*.—There is frequently a definite sensation experienced by the patient prior to the onset of the fit. This is spoken of as an *aura*. It may warn the patient of the oncoming fit and may enable him to lie down or take other precautions against hurting himself when the sudden unconsciousness ensues. The aura varies in nature, but it is oftenest some form of ordinary sensation, such as tingling, numbness and uneasiness. It affects the epigastric region most frequently, but may occur.

in any part of the abdomen, chest or extremities. The aura may be a special sensation, such as flashes of light or colour, or the hearing of sounds, or it may be psychical in character, viz., a peculiar dreamy state, a sensation of something wrong or of terror. A peculiar movement instead of a sensation may provide the warning, e.g., certain muscles may be thrown into contraction. Where the aura in successive attacks always affects the same place, e.g., the fingers or the toes, it may point to some organic lesion in the cerebral cortex presiding over this part. (2) *During the fit.*—The patient suddenly becomes unconscious and falls wherever he happens to be. He often hurts himself against hard objects or falls into the fire or into water. He commonly emits a loud cry just before falling, of which he himself is usually unconscious. He lies with his head thrown back and somewhat to one side, his legs outstretched, his arms usually flexed at the elbows and wrists and his hands clenched. All his muscles are in a state of *tonic* spasm, hence his breathing is impeded and his face, at first pale, soon becomes livid. This *tonic* stage generally lasts only a few seconds and is followed by the *clonic* stage, in which the muscular contractions become intermittent, fine in character at first, but gradually becoming coarser, so that the terminal clonic spasms are the most powerful. The eyes roll, the eyelids and the jaws open and shut, and the tongue is apt to be bitten severely if it is protruded. Froth, often blood-stained, comes from the mouth. The limbs may be jerked violently. The breathing is stertorous and the face becomes less livid. The urine is often evacuated, the fæces less frequently. (3) *After the fit.*—The clonic spasms generally cease after two or three minutes and the patient passes into a stage of *sleep or drowsiness* which lasts for two or three hours. Unconsciousness is usually deep at first, but lessens later, so that he can be aroused. When he awakes he may feel as well as ever or have some headache or confusion of ideas. The reflexes

are sometimes lost for a time and increased afterwards. The quantity of urine is often increased and there may be a trace of albumin or sugar. The epileptic fits may occur at night only and the patient be unaware of them for years. In exceptional cases either the *status epilepticus* comes on or a *trance-like* or *maniacal* state supervenes in which the patient may show homicidal or suicidal tendencies and perform acts of which he has afterwards no recollection. In the status epilepticus, fit succeeds fit, the patient remaining unconscious the while. The pulse and respirations increase and the temperature rises and death may follow from exhaustion.

2. The PETIT MAL or MINOR EPILEPSY. — It consists in a brief unconsciousness without convulsions. The patient stops suddenly in whatever he is doing, e.g., talking, eating, walking, &c., his face is pale, his eyes are fixed, there may be cessation of breathing, and anything held in the hand may be dropped. In a moment he resumes what he was formerly doing, entirely oblivious of what has happened. An aura may, but does not usually, precede these minor fits. The pupils often dilate, or alternately dilate and contract, not only during but for a time after the fits. The bladder may be emptied, especially by females, during a fit, but the tongue is never bitten nor does a drowsy state follow upon the fit. Giddiness is very frequently present during the fit and the patient often performs some almost automatic action, such as commencing to undress, or he may commit some strange or even illegal act. It is common for patients to exhibit the grand mal only, and some show the petit mal alone, while many others suffer from both. When the petit mal is present alone it is generally followed in the course of time by attacks of grand mal. The frequency of attacks vary greatly. A common interval is two or three weeks, but wide extremes are experienced. There may be only one seizure in a number of years or several attacks in one day. The attacks may come on singly or in groups.

3. JACKSONIAN EPILEPSY.—In this form the spasms begin in certain groups of muscles and spread to others, often becoming general. The localised form may exist alone for years, and when it ultimately passes into the general it usually does so rapidly, but may do so slowly. If the convulsions remain local, consciousness is preserved, if they become general it is usually lost. There may be a numbness or tingling localised to the part first affected. This form of epilepsy has already been referred to as likely to occur in cortical irritations, such as tumors or hæmorrhages, *vide* page 838. It also occurs in uræmia and in general paralysis of the insane.

Diagnosis.—The attacks of grand mal are so characteristic that there is mostly little difficulty in recognising them. Their simulation by hysterical patients may lead to mistakes. The most important distinctions are in hysteria, the greater duration of the attacks, the tongue not being bitten, though the lips, hands, or other people may be, the emotional character of the patient, the absence of enuresis, the frequently gradual onset of the attacks, and the way in which they succeed each other at intervals. Nocturnal epilepsy may be discovered by the patient complaining in the morning of headache, confusion of ideas, a bitten tongue and enuresis. The petit mal may be difficult to recognise. It may be simulated by attacks of syncope, of indigestion, or of Meniere's disease, from which it may be distinguished by attention to the state of consciousness and other conditions.

Prognosis.—Arrest is most likely when the fits begin after the age of thirty-five and when treatment is commenced before the fits have gone on for a year. Cases beginning between the twentieth and thirtieth years or under ten years of age give few arrests, whereas those

beginning at puberty give many. The outlook is better in males. The more frequent the attacks the graver is the outlook. Cases with marked mental disturbance are also unfavourable. Idiocy, mania, delusional insanity and homicidal and suicidal tendencies may follow. Death rarely occurs during a fit, but the patient may fall into the fire or water, or otherwise receive fatal injuries.

Treatment.—The first duty of the physician is to discover the causes of the epileptic seizure, or anything associated with it which might suggest a possible causal factor. Sometimes the *aura* indicates a definite part of the brain which is being irritated, and the removal of spicules of bone or a tumor pressing on the brain have been known to arrest the disease. Unfortunately it is only in a small number of cases that radical treatment is possible. Where the patient is a child, he should be carefully trained under conditions which obviate his being teased by other children, and in which his disability will not always be impressed upon his mind. There is no question that epileptic colonies and schools are of great value for epileptic adults or children who cannot receive the attention possible for patients in more affluent circumstances. Plenty of healthy exercise, a sufficiency of good food, and complete abstinence from alcohol are important. On no consideration should excitement be encouraged. In many cases it is desirable to teach the patient to use any information given him by the appearance of an *aura* that a fit is imminent. As a result the patient sits down, or lies down, and so escapes the risks of a severe fall. Where the fits occur during the menstrual epoch in girls, special attention should be paid to the administration of bromides during these periods. On no consideration should epileptic patients be allowed to marry. The common family history in epileptic patients is a previous record of cases of epilepsy,

and there is no type of case in which marriage should be so strongly discouraged as in the epileptic. The bromides are the remedies which still hold the first place in treatment. We give a mixture of equal parts of sodium, potassium and ammonium bromide, and a dose of 20 to 40 grains may be taken three times a day, or one dose, possibly 40, or even 60 grains, may be administered at bedtime, and especially where the fits are nocturnal in their incidence. Some physicians prefer one bromide to another, and the strontium salt has been much praised, although its greater efficacy seems doubtful. The risks more or less inseparable from the administration of bromides are bromism, bromide acne, and, what is of greater moment, the intellectual dulness and loss of memory which inevitably follow a long continued course of these drugs. It is well to intermit bromides after a period of six or eight weeks, and unquestionably the use of arsenic prevents the appearance of acne. Epilepsy also tends in time to damage the intellectual powers, and it is difficult to apportion to the use of the bromides the proper amount of blame, but it is desirable in all cases of epilepsy requiring prolonged treatment to give for a period of weeks other drugs than bromides, and amongst these may be mentioned belladonna (of which 5 minims of the tincture may be ordered thrice daily), chloral (20 grains), and cannabis indica.

Many patients derive great benefit from a course of cod-liver oil and iron, and change of air and scene are probably more necessary in cases of epilepsy than in the non-epileptic.

The other remedies which have been administered for epilepsy make up a very extensive list. Among the more important of these are ergot, nitro-glycerin, borax, salts of silver, zinc and other metals, and antipyrine. Occasionally opium, or one of its alkaloids, has been found of value, but its administration must be kept under careful supervision.

There are now many epileptic colonies scattered over the world, and most of these have been constructed on the lines originally followed by the first colonies established on the Continent. Two situated in England might be mentioned, viz., Chalfont St. Peters in Buckinghamshire, and Maghull Colony near Liverpool.

V. ECLAMPSIA—INFANTILE CONVULSIONS.

DEFINITION—Convulsive seizures in children or adults, closely allied to epilepsy, but differing therefrom in there being no tendency to many attacks and in disappearance on the removal of the cause. They may pass into true epilepsy, but do not do so necessarily.

Etiology.—The attacks are most common in young children. Certain influences may be regarded as causes, *e.g.*, instability of the nervous system and general debility. Rickets stands high among the causes of debility and is thus often associated with convulsions. Wasting diseases, such as long-continued enteritis or diarrhoea, so commonly caused in infants by unsuitable food, probably act in the same way, though they may also act more directly as exciting causes by reflex irritation. This latter action is well seen in the irritation of the first dentition, of a tight prepuce, of a foreign body, or of inflammation in the ear. Toxic causes are illustrated by the convulsions which often usher in infectious fevers, such as whooping-cough, measles, pneumonia and scarlet fever.

Symptoms.—Twitchings, movements of the mouth or hands, often associated with grinding of the teeth, are usually the first symptoms. General convulsive movements follow or may exceptionally come on without any previous symptoms. They are not so complete as in true epilepsy, but there is definite tonic spasm, causing the body to become

rigid and the face blue and cyanosed. Clonic spasms follow, the face, arm, and leg muscles twitch, the eyes roll and the head is retracted. The attack gradually subsides and is followed by drowsiness. There may be only one attack, but there is a tendency to recurrence, and this may happen with such frequency and rapidity that death follows from exhaustion. The attacks disappear with the removal of the cause.

Treatment.—The cause must be sought for and removed, and in many cases attention to the state of the mouth and stomach yields indication of what requires to be done. Lancing the inflamed gum or giving an emetic often proves most successful. The dietary requires study, and where the fits come on in wasting diseases this is of the greatest moment. Often most unsuitable food is being given, and this must be stopped. Chloroform anæsthesia arrests repeated and severe fits, but as a rule a hot bath is sufficient. Bromides in doses of 5 to 10 grains twice or thrice daily often control the attacks in children.

VI. OCCUPATION NEUROSES—PROFESSIONAL SPASMS.

Syn. *Writer's Cramp or Scrivener's Palsy, Telegraphist's Cramp.*

DEFINITION—A group of nervous affections incidental to various occupations, *e.g.*, writing, piano-playing, telegraphy, cigarette making, weaving, &c., characterised by irregular spasm or cramp in the muscles ordinarily in use which interferes with the performance of the action.

Etiology.—An excessive or improper use of the muscles concerned in the action. Writer's cramp is the commonest form. It occurs more frequently in men than women. Gowers states that a faulty method of writing, using the little finger or wrist as the fixed point instead of the middle

of the forearm or elbow, has been followed in the majority of cases.

Morbid Anatomy.—Definite changes in the motor nerve cells have rarely if ever been found. Wasting or paralysis of certain muscles is sometimes present.

Symptoms.—There are disturbances of co-ordination, spasms, paralyses, tremor, pain and trophic changes. The spasms most commonly affect the forefinger and thumb, interfering with the delicate movements necessary in writing, and sometimes affecting other muscles near, causing the pen to execute violent movements or to be jerked out of the hand. The paralyses are slight and affect only the muscles which hold the pen. Though they feel weak the grasp of the hand may be strong. They are often present along with the spasms. The tremor is seen in the affected muscles. Pain is rare, but numbness, soreness and tingling or tiredness are common. The trophic changes are rare, the fingers becoming glossy and subject to chilblains. Electrical irritability is often diminished in the affected muscles.

Diagnosis.—It is generally obvious. Nervous people may imagine that the disease is coming on, but they may be reassured by the absence of the characteristic signs. Miner's nystagmus is closely allied to these trade spasms.

Prognosis.—The disease runs a slow course. Rest may be followed by considerable improvement, but the outlook is unfavourable, for the disease is apt to return and to appear also in the other hand if the patient educates himself to write with it.

Treatment.—From what is written above, rest is the chief method of treatment, and the cessation from the

movements in question should be absolute and prolonged. Typewriting and various devices, such as writing from the shoulder, are recommended, but all methods of writing should be used with care. Massage and rhythmic exercises and galvanism are helpful in certain cases. Above all, the health of the individual should be improved by such tonics as iron, arsenic, and phosphorus.

VII. TETANY.

DEFINITION.—Tonic spasms affecting chiefly the extremities.

Etiology.—It affects both children and young adults. Debilitated and toxic conditions are present in many cases. Gastric and intestinal disorders, rickets and fevers are examples of debilitating causes often followed by tetany. Poisoning by chloroform, morphia, lead, alcohol, &c., the infectious processes, *e.g.*, typhoid, cholera, influenza, &c., as well as thyroidectomy, including the removal of the parathyroids, are similar illustrations of toxic influences. The disease, generally an acute form lasting two or three weeks, may occur as an epidemic.

Morbid Anatomy.—No morbid change has been discovered.

Symptoms.—Characteristic spasms appear in the hands and feet, often called carpo-pedal spasms. The fingers are flexed at the metacarpo-phalangeal joints, extended at the interphalangeal joints, and the thumb is adducted and may be flexed in the palm of the hand. The wrist and the elbow are often slightly flexed, the foot is extended at the ankle and strongly arched. The toes are adducted and flexed. The muscles of the face and neck are less

commonly affected, but in severe cases there may be some trismus. The attacks set in suddenly and are mostly paroxysmal, the spasms lasting for minutes or hours, and being often painful. Occasionally they are continuous and persist for two weeks or more. Sleep diminishes but does not always abolish the spasms. The intervals between the spasms may last for hours or days, and the spasms may be induced by either pressing the nerve trunks or the blood-vessels of the part (Trousseau's symptom) or by tapping the affected muscles (Chvostek's symptom). The electric reactions are often greatly increased.

Diagnosis.—The history, course and character of the spasms permit of an easy diagnosis in most cases.

Prognosis.—Most cases recover within a few weeks. Death is rare even in the acute cases, though it has occurred in cases following acute dilatation of the stomach and also thyroidectomy.

Treatment.—There is almost always a definite cause which must be searched for and treated. In thyroidectomy cases the patient should at once be put on thyroid extract, and, where there is a rachitic element present, the child should be given fresh milk with plenty of fatty matter. The attack generally yields readily to hot baths, with sometimes cold shower baths added. Electricity, especially galvanism, is helpful. Chloroform can be administered where the attacks are very severe, but bromides graduated to suit the patient's age are more suitable antispasmodics. Where a dilated stomach is the cause of the tetany posterior gastro-enterostomy is indicated, and especially if a brief trial of lavage has proved unsatisfactory.

VIII. PARAMYOCLONUS MULTIPLEX— MYOCLONIA.

This is a rare disease characterised by clonic contractions, sometimes constant, sometimes paroxysmal, of certain muscles of the limbs. The movements are sometimes very violent. Male sex, heredity and fright all seem to play a part in the causation of the disease, but very little is really known about it.

IX. MYASTHENIA GRAVIS.

This is a rare disease resembling bulbar paralysis in its symptoms but showing no recognisable morbid change. Campbell and Bramwell have collected and analysed about sixty cases. It chiefly affects young people, and exposure to damp and over-exertion seem to predispose to it. It consists in the rapid exhaustion of certain muscles, first those of the neck, face and mastication, but later it may be all the voluntary muscles, by work and faradic stimulation. After rest the power is recovered. This rapid exhaustion of the muscles by faradism, not by galvanism, is called the myasthenic reaction. It, the ptosis and nasal speech, along with the absence of atrophy in the affected muscles, make the diagnosis easy. It is a very chronic disease. About a third of the cases were fatal and many recovered.

Treatment. — Very little can be done as a rule, but certainly strychnine should be administered, and potassium iodide and mercury have been recommended.

X. MIGRAINE—HEMICRANIA—SICK HEAD-ACHE.

DEFINITION—Paroxysmal headache, usually unilateral, often associated with sickness and disturbances of vision.

Etiology.—Heredity, age, sex, reflex irritation, mental and physical fatigue all seem to have some influence in its production. It has occurred in families through several generations. It is commonest in women, particularly of a neurotic tendency, and mostly comes on early in life. Reflex irritation of some kind is present in many cases. The trouble may be in the nose, teeth, throat, stomach, intestines, ovaries or uterus, or in the eyes.

Morbid Anatomy and Pathology.—There is no definite lesion, but various theories have been advanced. A neuralgia of the first division of the fifth nerve has been suggested, but the long continuance of the disease and the lengthened intermissions are against it. Stimulation followed by exhaustion of the sympathetic, producing first spasm and then dilatation of the blood-vessels, has been thought to explain the attacks, but the experience of many, *e.g.*, Lauder Brunton in his own case, shows that the state of the blood-vessels may vary in different attacks in the same person. Liveing advanced another theory that the attack is of the nature of a "nerve storm," due to a temporary disturbance of function in certain cerebral nerve centres.

Symptoms.—In most cases some premonitory signs precede the headache, such as malaise, depression, peculiar vision disturbances, *viz.*, zigzag lines, flashes of light, dimness of vision, or hemianopia. They are quickly followed by the headache which begins in one temple or other part and rapidly increases in intensity and extent until it involves the entire side of the head and sometimes both sides and

the neck. It is boring, throbbing and expansile in character. It lasts for a variable time, one or more hours or a day, occasionally for several days, generally prostrating the patient, and often passes off with nausea and vomiting or sleep. The attacks may recur for many years but tend to cease after middle life. They do not endanger life.

Treatment.—However difficult it may be to decide what causes the attack of pain, an effort should be made to exclude all conditions which may act as possible exciting or predisposing factors. The sight must be thought of, and especially any astigmatism should be investigated and if any error is found it should be corrected with suitable glasses. The nose, the ears, the pelvic organs in women, and the teeth must all receive attention.

Errors of diet, disturbances of stomach and bowels should be put right, and the important question of the patient's work, or rather overwork, should be considered. In many cases a holiday is the best remedy, and there must be no overstrain of any of the faculties.

Some cases seem to be associated with a too sedentary life, and exercise obviates or lessens the severity of the attack. The relief of the headache is often attained by inducing emesis and then giving one or other of the coal-tar group of remedies. Phenacetin, ammonol and phenalgin are prompt in action. One or other of these remedies will arrest the headache if the stomach is not full and the drug gets a chance of being absorbed. Many patients find relief from taking a 3 to 6 grain blue pill at bedtime, followed up with a saline purge in the morning. Among the most rational suggestions is that of Herter, who advises lavage at once when an attack threatens, and then to administer a saline purge. Sometimes there is high arterial pressure in the carotid, if so, nitrite of amyl may be tried, or a course of nitrite of soda (2 to 4 grains thrice daily).

XI. HYSTERIA.

DEFINITION—A very common disease showing a great variety of sensory, motor, vasomotor and psychic symptoms assignable to instability of the nervous system and lack of will power and not to any known organic disease.

Etiology.—*Sex and age.*—It is more common in women, particularly about the time of puberty, but is not uncommon in boys and also occurs in men. It begins most frequently from fifteen to twenty years of age, though it may do so quite early or late. No race is exempt from it, but the Latin races are the most susceptible to it. *Heredity.*—There is often a history of insanity, epilepsy or other neurosis in the family. *Debility.*—Any long-continued ill-health, *e.g.*, syphilis, tubercle or wasting disease or a weak constitution, may help to bring on the disease, particularly in patients susceptible to it. *Faulty education*, both at home and at school, *e.g.*, overwork and over-indulgence, has an important influence; the weakly, spoilt child is apt to become hysterical. *Violent emotions*, *e.g.*, frights, love affairs, bereavements, family and business worries, religious difficulties, often seem directly associated with its onset. *Sexual affairs*, *e.g.*, masturbation, sexual excess, precocious interest in sexual matters, at times actual disease of the sexual organs, particularly when they require prolonged treatment, are weighty influences. *Physical injury* appears to start some cases. *Imitation* has occasionally been responsible for an epidemic in a school, nunnery, or a village.

Morbid Anatomy.—There is no known change associated with the disease. It is probable that in many cases there are minute though as yet unrecognised morbid changes in some of the higher nerve-cells, and in some cases there are actual organic lesions present in the body which may influence in some measure the course and character of the

disease, but never do so to such an extent that the symptoms can all, or mainly be directly assignable to them.

Symptoms.—They may be usefully divided into those which are paroxysmal or convulsive and those which are not, though the convulsive symptoms are absent in many cases.

Paroxysmal Symptoms.—Hysterical fits generally begin after some special emotion. They vary greatly in severity from a mere exhibition of excitement or swooning, in which the patient does not lose consciousness, to a severe epileptiform attack with a complete loss of consciousness. They may begin suddenly without warning or be preceded by uneasiness, palpitation, a sense of choking or of a ball rising in the throat (*globus hystericus*), or faintness. The patient indulges in a fit of loud laughter or sobbing or both, sinks into a chair or on to the ground, tosses her arms about, swoons or exhibits other signs of loss of control. The fit may last only a minute or two or much longer. In many cases it begins more suddenly and is more severe. The patient may fall with or without a cry, like to but not so suddenly as true epilepsy, generally selecting a suitable spot, chair or bed, and often a suitable audience. General clonic spasms appear, often preceded by tonic spasms, which may, indeed, prevail throughout, she tosses her arms widely, struggles, throws herself about but does not hurt herself much, never bites her tongue, though she may bite her hands or injure persons near her. She has the appearance of being not entirely unconscious, and often talks of those about her, and though sympathy makes her worse, she is probably unconscious or nearly so during the greater part of the attack. It may last only for a few minutes or be considerably prolonged and be followed by other similar fits at short intervals without the corresponding gravity of the status epilepticus. The fit frequently ends in an outbreak of crying and is generally followed by the evacuation of a large quantity of pale urine of low specific gravity. There is no drowsiness

after the fit, though there may be a semi-torpid state. In the most severe form, hystero-epilepsy, or the *grande hystérie* of the French, which is rare in this country, the attack still more closely resembles true epilepsy, from which however it is generally to be distinguished by the non-biting of the tongue, the non-occurrence of involuntary micturition or defæcation, or by the result of pressure in the inguinal region or other hystero-genetic area (certain sensitive skin areas in the front and back of the trunk), which may either stop or bring on an attack. The spasm stage may be followed by contortions and cataleptic poses (the clownism of Charcot), then by attitudes and facial expressions indicating intense love, pain, hate or other passion, and lastly by a stage of delirium or hallucinations which she may remember more or less fully after recovering.

Inter-paroxysmal symptoms.—These are the more or less permanent symptoms of the disease. They are extremely variable and are best considered under the systems to which they belong.

1. *The sensory symptoms.*—Anæsthesia is very common, it may involve the whole of one side, including the special senses, or certain spots or segments. In mild cases it involves only the skin, but in others it involves also the mucous membranes, *e.g.*, the soft palate, and the deeper parts. It may affect painful and thermic sensations and the muscular sense as well as touch. Hyperæsthesia in the form of pain or tenderness in certain areas in the skin or joints is common. Disease of the joints is simulated. The least movement causes pain and the joint is fixed; it is often swollen, and in prolonged cases it looks more enlarged than it is, owing to the wasting of the muscles.

Tenderness is commoner than spontaneous pain. The hystero-genetic areas above mentioned are hyperæsthetic. The pain may be moved from one place to another by magnets and other devices, no doubt due to "suggestion." Sight may be affected, one or more blind spots may be

present in the visual field, or blindness may be complete. Colour vision may be affected. The other special senses are also implicated. All these phenomena are temporary, though they may last long and return.

2. *Motor Symptoms.*—They are common and striking features in the disease. Almost every possible form of motor disturbance met with in organic diseases and many not met with may be experienced. Walking, sitting, standing, balancing, any or every form of muscular movement may be impaired. Locomotor ataxia or cerebellar disease may be simulated. True paralysis, either flaccid or spastic in type, hemiplegia, paraplegia or monoplegia may be mimicked. They are to be distinguished chiefly by the absence of wasting in the muscles, or by the paralysis involving all the muscles equally and not special muscles, and by its rarely being extreme, and by the absence of the reaction of degeneration. The tendon reflexes are either normal or increased. Aphonia is often present. Contractures are common and may be extreme. The nails may be driven into the palm of the hand, the leg may feel like an iron bar, the abdominal muscles may simulate a phantom tumor, the mouth may show trismus. These contractures do not resolve during sleep and often not until full anæsthesia is produced. Rhythmic spasms may occur, *e.g.*, salaam or choreiform movements, also tremors of many kinds.

3. *Visceral Symptoms.*—*The alimentary system.*—The digestive functions are often altered. There may be globus hystericus, dysphagia, vomiting, loss of, or capricious appetite, ptyalism, diarrhoea, and noisy eructations. Nutrition is rarely affected in proportion to the severity of the apparent loss of appetite and amount of food taken, since the patient often eats in secret; but in the severe form, anorexia nervosa, appetite is completely lost, vomiting is frequent, and the wasting extreme. *The circulatory system.*—Palpitation and cardiac pains are common. *The respiratory system.*—

A loud barking or dry irritating cough is a prominent feature. Cries of various kinds, imitative of animals, hiccup, yawning and sneezing fits may be noticed. Aphonia is common. The patient speaks in a whisper, though the cough may be as loud as natural. It may appear and disappear suddenly. Laryngoscopic examination is easy owing to the anæsthesia of the palate. *The genito-urinary system.*—Retention of urine is common. It is apt to appear and disappear irregularly and may need catheterisation, though this should be avoided as far as possible. Polyuria after the hysterical fits is usual.

4. *Mental symptoms.*—Lack of will control and intense desire for sympathy are the leading mental characteristics of the disease. The moral sense becomes perverted and frequently no reliance can be placed upon the patient's statements. Substances may be added to the urine, foreign bodies inserted into the body passages, soap into the mouth to cause frothing, or other deceitful acts committed with the object of suggesting the presence of real disease. Delirium may be assumed, and loathsome animals imagined to be visible or existent within the body. Mental depression is common, and the cases may simulate hypochondriasis or insanity, into which they sometimes pass. There may be inability to stand (astasia) or to walk (abasia), or a state of catalepsy (in which the limbs remain for a long time in any position in which they are placed) or trance (a profound death-like sleep, lasting for hours, days or months) may ensue. The temperature may rise in a remarkable manner (the hysterical fever), particularly in severe attacks, records of 109°F. and 118°F. having been obtained. These high temperatures are often due to fraud, but others of them have defied all explanation.

Diagnosis.—Many cases are easy enough and, in the more difficult ones, attention to the nervous state of the patient, to the characters of the seizures, to spontaneous

disappearance and reappearance of symptoms, to the effect of faradism and deep anæsthesia and the absence of proof of the existence of organic lesions, will generally enable a diagnosis to be made.

Treatment.—There is no disease which is so largely the result of faulty upbringing and education but much can be accomplished by teaching self-control and preventing excessive brain work in obviating the onset of hysteria in young persons who are of neurotic tendencies. When the disease has developed the treatment may be divided into (1) the general management of the case, (2) the measures used during an attack of hystero-epilepsy, or the *grande hystérie* referred to above, and (3) the treatment of special symptoms.

The general management implies the Weir-Mitchell method of placing the patient under the care of kind but strong-minded attendants, and if possible away from home and friends. The diet should be generous and in some cases excessive, forced feeding is of value while massage is being regularly carried out. The patient should be interested in other things than her own ailments, and after a period of rest and full dieting, improvement in any symptom from which she suffers should be rewarded by some prize—in fact she should be treated much as one would treat a sick and fractious child. Valerian and asafoetida are useful remedies in some cases, and musk has obtained considerable notoriety, although the benefit obtained from it in our hands has not been striking.

Should a hysterical fit occur, electricity, cold shower baths and similar measures are often of value in arresting the attack and deterring future seizures, because although once started the patient has no control over the fit, unquestionably in not a few cases by an effort the fit can be stopped altogether. As little attention as possible should be paid to the patient during the actual seizure. Electricity is often

of use for symptoms of different kinds, and especially for aphonia, paralyses of muscles of limbs, and so forth, sometimes complete recovery suddenly taking place. As a general method of treatment static electricity is beneficial. Much may be done by judicious suggestion and cultivating will power, but the practice of hypnotism is to be discouraged. Pain, and in fact any hysterical ailment which may arise, should never be treated with any opiate such as morphia, although the bromides are often helpful if used to allay unusual excitement. An occasional fly-blister or the application of the button cautery relieves the pain common over one or other hystero-genetic area.

XII. NEURASTHENIA.

DEFINITION.—A condition of exhaustion of the nervous system causing both physical and mental irritability and inefficiency.

Etiology.—*Predisposing causes* are hereditary influences (parents transmitting feeble mental organisation to their offspring) faulty education, general ill-health, and disturbed sexual passion. The *exciting causes* are overwork and worry, infectious diseases, the drug habit, dissipation, religious excitement and similar influences. Injury is sometimes an exciting cause. Of all these, strain and worry are the most common and their effects are often seen in business and professional men who have stuck too closely to their work.

Symptoms.—The symptoms are extremely varied, but an attempt may be made to arrange them under similar headings to those of hysteria, viz., sensory, motor, visceral and mental. The *sensory symptoms* are perhaps the commonest. Hyperæsthesia is generally marked and refers mainly to sensations of pain and a sense of pressure. The pain is often much complained about. It

disappearance and reappearance of symptoms, to the effect of faradism and deep anæsthesia and the absence of proof of the existence of organic lesions, will generally enable a diagnosis to be made.

Treatment.—There is no disease which is so largely the result of faulty upbringing and education but much can be accomplished by teaching self-control and preventing excessive brain work in obviating the onset of hysteria in young persons who are of neurotic tendencies. When the disease has developed the treatment may be divided into (1) the general management of the case, (2) the measures used during an attack of hysterо-epilepsy, or the *grande hystérie* referred to above, and (3) the treatment of special symptoms.

The general management implies the Weir-Mitchell method of placing the patient under the care of kind but strong-minded attendants, and if possible away from home and friends. The diet should be generous and in some cases excessive, forced feeding is of value while massage is being regularly carried out. The patient should be interested in other things than her own ailments, and after a period of rest and full dieting, improvement in any symptom from which she suffers should be rewarded by some prize—in fact she should be treated much as one would treat a sick and fractious child. Valerian and asafoetida are useful remedies in some cases, and musk has obtained considerable notoriety, although the benefit obtained from it in our hands has not been striking.

Should a hysterical fit occur, electricity, cold shower baths and similar measures are often of value in arresting the attack and deterring future seizures, because although once started the patient has no control over the fit, unquestionably in not a few cases by an effort the fit can be stopped altogether. As little attention as possible should be paid to the patient during the actual seizure. Electricity is often

of use for symptoms of different kinds, and especially for aphonia, paralyses of muscles of limbs, and so forth, sometimes complete recovery suddenly taking place. As a general method of treatment static electricity is beneficial. Much may be done by judicious suggestion and cultivating will power, but the practice of hypnotism is to be discouraged. Pain, and in fact any hysterical ailment which may arise, should never be treated with any opiate such as morphia, although the bromides are often helpful if used to allay unusual excitement. An occasional fly-blister or the application of the button cautery relieves the pain common over one or other hysterogenetic area.

XII. NEURASTHENIA.

DEFINITION—A condition of exhaustion of the nervous system causing both physical and mental irritability and inefficiency.

Etiology.—*Predisposing causes* are hereditary influences (parents transmitting feeble mental organisation to their offspring) faulty education, general ill-health, and disturbed sexual passion. The *exciting causes* are overwork and worry, infectious diseases, the drug habit, dissipation, religious excitement and similar influences. Injury is sometimes an exciting cause. Of all these, strain and worry are the most common and their effects are often seen in business and professional men who have stuck too closely to their work.

Symptoms.—The symptoms are extremely varied, but an attempt may be made to arrange them under similar headings to those of hysteria, viz., sensory, motor, visceral and mental. The *sensory symptoms* are perhaps the commonest. Hyperæsthesia is generally marked and refers mainly to sensations of pain and a sense of pressure. The pain is often much complained about. It

may affect the head, the eyes, the joints, the back, or the viscera. Pain or a sense of weight or oppression on the top of the head is frequent. There may be certain painful spots along the spine or elsewhere. Feelings of tiredness, numbness and tingling occur. The special senses may be disturbed. There may be flashes of light or an aching in the eyes ("the irritable eye") after any effort of sight. The patient may complain of unpleasant smells emanating from his body. *Motor symptoms* are not common, but there may be great muscular weakness. Restlessness is not unusual. Later on, the muscular weakness or general debility causes the patient to be confined to bed. *Visceral symptoms*.—Palpitation, quick action of the heart, dizziness, a throbbing or jumping abdominal aorta suggestive of aneurism, flushes of heat in the head, flushing of the skin, profuse general or local perspiration, coldness of the extremities are liable to occur. A painful testicle or ovary, spermatorrhœa, nocturnal emissions or a dread of impotence may be complained of. The mental symptoms dominate most cases. There is always a loss of the power to concentrate the attention. The memory suffers. The patient becomes introspective and engrossed in his troubles. He becomes moody, indecisive and depressed. Sleeplessness is common. He is often anxious, greatly disturbed by his own sensations, is oppressed by various fears or "phobias," *e.g.*, agoraphobia, *i.e.*, fear of open spaces, claustrophobia, *i.e.*, fear of being left alone in a small room or place, anthropophobia, *i.e.*, fear of people, and many others. The mental distress may be so great that the patient cannot rest or sleep but tosses about, crying, complaining and despairing, and may resort to suicide.

Diagnosis.—The absence of organic disease, the disproportion between the patient's subjective distress and any

ascertainable cause, the history and the mental condition of the patient make the diagnosis clear in many cases. Cases which run into hypochondriasis and hysteria are the most difficult. Some cases resemble tabes dorsalis and others general paralysis, but a careful examination of the symptoms will generally suffice to distinguish them.

Prognosis.—The outlook is often good, particularly under proper treatment. Relapses are common.

Treatment.—The first thing to be done if the case is seen at an early stage is to make the patient take a complete rest—physical and mental. Where the symptoms demand it, a prolonged period of quiet confinement in bed, with massage and a simple but full dietary, will often effect a cure, but in less marked cases a holiday is sufficient. Occasionally where overwork mentally has caused the breakdown, a holiday with plenty of physical effort but no brain excitement is indicated, but this is by no means suitable for the majority of patients.

Self-control must often be inculcated, and the judicious physician can reassure a nervous patient and train the sufferer to disregard his more imaginary symptoms, while interesting him in a suitable occupation which does not imply brain effort. The Weir-Mitchell plan of treatment is sometimes necessary, and certainly massage and electricity in the form of either static or high-frequency applications are of benefit. Where there are areas of pain, local fly-blisters may do good, but drugs other than tonics are not desirable, although sometimes severe neuralgic pain demands an occasional dose of phenacetin or similar remedy. The best tonics are iron, arsenic, strychnine and cod-liver oil. Sleeplessness should not be treated by hypnotics unless in quite exceptional circumstances, and no such drug should be taken by the patient without the knowledge and approval of the medical attendant.

At a later period cold baths or shower baths may help convalescence, together with gradually increasing physical exercise, but what may suit one patient will not suit another, and every patient's case requires careful study. A gain in weight of one or two pounds a week is hopeful and indicates that the treatment adopted is succeeding. Baths have been much recommended, by which we mean not only the change of scene and association of a Spa, but the various kinds of hydropathic treatment practised at most of such baths so far as they are likely to act as sedatives or mild tonics, or both combined.

XIII. TRAUMATIC NEUROSES.

DEFINITION.—A group of affections following shock, known as railway brain, railway spine, traumatic neurasthenia, traumatic hysteria.

Etiology.—Injury or shock, particularly from railway accidents.

Symptoms.—The physical injury may appear to be slight, particularly at the time, and the patient may be able to resume his ordinary work, but after a few days or weeks neurasthenic or hysterical symptoms appear. In other cases the symptoms suggest organic disease of the brain or cord, sensory, motor, visceral and mental symptoms occurring in varying combinations, and death sometimes supervening, though no actual lesion may be discovered during life or after death.

Diagnosis.—The history and a careful study of the case may make the diagnosis easy. The question of the presence or absence of organic disease has always to be most care-

fully determined, and in some cases it must remain in doubt, at any rate for a time.

Prognosis.—The outlook is often good, particularly after a favourable verdict has been obtained in the law courts; but it should always be remembered that cases in which no trace of organic lesion has been found may go steadily downwards and ultimately prove fatal.

Treatment.—What has previously been stated with regard to neurasthenia is applicable here.

VASOMOTOR AND TROPHIC DISEASES.

I. ANGIO-NEUROTIC ŒDEMA.

DEFINITION—An affection characterised by transient local œdema, often associated with colic and showing a hereditary tendency.

Etiology.—It is apparently a vasomotor disturbance and it may be associated with other like manifestations, such as urticaria, purpura, Raynaud's disease, but beyond a tendency to heredity nothing is known as to why these disturbances should appear.

Symptoms.—Local, usually circumscribed, areas of œdema appear on any part of the body. The face and eyelids are common situations and the swelling may be so great as to prevent the eyelids or the mouth being opened. Other situations are the backs of the hands, the larynx, the legs, the genitals and indeed any part of the body. The most striking feature about these attacks of œdema is that they are *transient*, lasting for not more than two or three hours as a rule, and that they tend to *recur* every few weeks or months and sometimes at definite periods, the patient

being perfectly well between the attacks. During the attacks there is little suffering other than that induced by the swelling—though when this involves the larynx it may prove fatal (œdema glottidis)—except some gastro-intestinal disturbance. This may be slight or severe. Colic is its most frequent form, and its severity may be so great as to necessitate the use of morphine. At other times there is nausea or vomiting. There is frequently a history of the disease in several members of the family throughout several generations.

Treatment.—Tonics such as iron and arsenic, and rest in bed in severe cases, often prove sufficient. Tracheotomy may be called for if the œdema is laryngeal. Any other element in the case, such as the presence of purpura in Raynaud's disease, may be helpful in suggesting suitable treatment.

II. RAYNAUD'S DISEASE.

Syn. Symmetrical Gangrene, Local Asphyxia.

DEFINITION—Local symmetrical asphyxia often followed by gangrene, probably due to a combination of vasomotor spasm and paresis.

Etiology.—It is more common in women, particularly those of a neurotic constitution. A hereditary history of it is sometimes obtained. Cold and fatigue are the two causes most likely to bring it on in susceptible persons.

Morbid Anatomy.—There is strong evidence that spasm of the arteries and arterioles exist along with dilatation of the capillaries and small veins. The nerves may show some peripheral neuritis.

Symptoms.—The fingers, toes and ears are the parts affected, the fingers most commonly of all. The earliest

stage is a numbness and bloodlessness of one or more fingers, exactly similar to that produced by cold, a state known as *local syncope*. The fingers, often only their terminal parts, rarely the whole hand, become cold, dead white and numb, and after a short time, minutes to hours, red-hot and burning and gradually return to normal, a condition similar to that occurring in and familiar to children indulging overlong in snowballing. The affected fingers may not pass through the stages equally quickly or be equally involved, and consequently may exhibit different appearances at the same time. Cold, emotion and fatigue seem to favour attacks, which may recur at intervals, particularly in winter, during years without getting any worse. They often, however, pass into a further stage known as *local asphyxia*, which may come on independently. The fingers, toes, ears or nose, sometimes the whole hand, become swollen and intensely congested. They may feel burning hot or stone cold. Chilblains are common. Numbness or pain may be great. The attacks last a variable time and then pass away. In this stage vasomotor spasm of the arterioles probably exists along with paralysis of the capillaries and small veins. In a number of cases the attack persists until it causes death of some of the tissues. This is the third stage of *local gangrene*. It may involve very small parts of the tips only of the fingers, or the terminal phalanges or larger pieces, and even the whole hand, but severe gangrenes are rare. The parts implicated become gradually black and blebs may form in the skin. A line of demarcation slowly forms and there is a gradual loss of tissue from sloughing, but the extent of this loss is much less than one usually sees in other gangrenes. As a rule the losses are quite superficial, but a part or whole of one or more fingers, the tip of the nose and ear, or even a whole hand or part of the arm may be lost. Cases of extensive multiple gangrene are rare. They occur usually in children, and death may ensue in a few days. Numbness and tingling

may persist for long periods in parts which have recovered. Other skin and visceral disturbances may be associated with Raynaud's disease, *e.g.*, erythema, scleroderma, eye and nervous changes and paroxysmal hæmoglobinuria. Exposure to cold may bring on both an attack of Raynaud's disease and paroxysmal hæmoglobinuria. Occasionally sugar appears in the urine. The nervous changes are transient hemiplegia and great depression or more rarely mania.

Treatment.—In severe cases the patient must be guarded against cold and should be kept in bed during frosty weather. If able to do so, he should be ordered to a warmer climate during the more inclement months of the year. The affected limb or limbs should be wrapped up in cotton wool during an attack. Should the pain be very severe opium may be necessary. The element of arterial spasm suggests a protracted trial with the nitrite group of remedies, and $\frac{1}{100}$ th grain of nitro-glycerine thrice daily may be found helpful. Cushing has suggested constricting the affected limb with a tourniquet for a few minutes, and even repeating the procedure if the arterial spasm does not relax with the first application. We have seen galvanism of distinct service in one case, one pole being applied to the spine and the other immersed in salt water along with the affected limb.

In some cases a floating kidney has been found associated, and operative interference (nephrorraphy), as recommended by Dr G. A. Gibson, has proved most successful, apparently terminating the attacks of local syncope.

III. ERYTHROMELALGIA (RED NEURALGIA).

DEFINITION—A rare chronic form of neuralgia accompanied by redness and swelling.

Etiology.—Cold seems able to induce an attack in some people, but in a few other cases cold has relieved while heat made it worse.

Symptoms.—It affects the hands and feet, the latter more frequently. It may be one or both. The affected parts become painful, sometimes agonisingly so, then swollen and congested. The skin and nails may atrophy. It is occasionally complicated with Raynaud's disease.

Treatment.—The affected limbs should be protected from cold, and where exposure, as work in cold water, has apparently caused the affection, the patient should be guarded from continuing at employment which is hurtful. The hands if affected may be carried in slings. The diet should be generous.

IV. SCLERODERMA.

DEFINITION—A circumscribed or diffuse induration of the skin and subcutaneous tissues.

Etiology.—It affects women more than men, most commonly at the middle period of life. No cause is known, it is closely related to the tropho-neuroses.

Symptoms.—There is a circumscribed form in which there are varying sized patches, often small, brawny, hard and dense on the neck or chest, the favourite situations, sometimes along the course of the nerves. These are inelastic and difficult to move over the tissues beneath them. The skin covering them is either paler or more pigmented

than usual and secretes no sweat. They may appear rapidly, disappear as rapidly, or last for years. There is a diffuse form which is less common. It usually involves first the extremities or the face and may become general. In many cases it affects either the trunk or arms, in fewer cases the legs. The affected parts are fixed and hide-bound. The face is expressionless, the lips and fingers immobile. The skin is pale, smooth and dry and cannot be pinched up between the fingers. There is great increase in the fibrous tissues of the skin and subcutaneous tissues and sometimes an interstitial myositis. The disease is very chronic and may last for years. It may steadily progress or become arrested and disappear. It may be associated with disturbances of sensation and vasomotor control, and Raynaud's disease may be present. Lung or kidney trouble is apt to supervene.

Treatment.—Much may be done by systematic massage and applying elastic bandages. In diffuse cases thyroid extract should be tried, but it is not always of much or any service. Diet the patient and clothe warmly where there is sensitiveness to cold.

V. FACIAL HEMIATROPHY.

This is a rare disease in which all the tissues (bones and soft parts) of one side of the face, the upper part more than the lower, are atrophied. It usually begins in childhood and at one spot, from which it spreads. It is limited sharply to one side of the face, giving the patient a curious appearance. The skin and hair often change colour and the teeth fall out owing to the wasting of the alveolar processes. The corresponding half of the tongue usually atrophies. It is sometimes ascribed to an affection of the fifth nerve, but little is known about it.

VI. ACROMEGALY.

DEFINITION—A chronic affection characterised by great increase of growth in certain of the bones of the face and extremities.

Etiology.—It generally begins in early adult life and is more common in women. It may be related to hypertrophic osteo-arthritis and gigantism. Rheumatism, mental worry and syphilis have been thought to be connected in some way with its origin, but the connection is doubtful. It is sometimes looked upon as a nutritional disturbance due to disease of the pituitary body, which may be the growth centre of the skeleton.

Morbid Anatomy.—The pituitary body has been altered in most cases, either hypertrophic, cystic or the seat of a tumor. Both the thyroid and thymus have been enlarged in some cases, atrophied or normal in others. The affected bones are hypertrophied, the new growth being subperiosteal.

Symptoms.—The hands and feet are enlarged but not deformed and can be freely moved, the big toe being often proportionately larger than the others. The lines on the palms are deepened. The arms and legs escape in such cases. The head is enlarged, particularly the face, the increase in size of both upper and lower maxillæ being great, the latter being more marked than the former, so that it often projects below the upper jaw. The spaces between the teeth are increased. This is regarded as an important early sign. The nostrils are large and broad, the eyelids are thickened and the ears enlarged. The muscles are sometimes atrophied. The gait becomes slow and slouching and kyphosis appears. Somnolence and dulling of the mental powers may occur. Menstrual disturbance may arise early in the disease: optic atrophy, more rarely

neuritis, may be present, and bitemporal hemianopia may set in early, due to pressure of pituitary body on optic chiasma.

Prognosis.—The disease is very chronic and not dangerous to life, as patients may live for fifteen, twenty or more years.

Treatment.—Pituitary extract and thyroid extract have both been used, although with only partial success in a very few cases.

VII. HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY.

There is an enlargement of the hands and feet and of the lower ends of the long bones, particularly of the forearm and legs, but the head and face are not involved. The terminal phalanges are much enlarged. It is very chronic and usually attacks adult males who have suffered from long standing pulmonary disease, of which chronic bronchitis, tubercle, sarcoma, empyema and asthma have been most frequent.

VIII. OSTEITIS-DEFORMANS—PAGET'S DISEASE.

This is a rare disease, comprising enlargement, softening and curving of the long bones, and enlargement of the head. There is a rarifying osteitis with some new formation of bone, so that the bones are much thicker though less resistant. The cranial bones may be increased several times in thickness, and the face assumes a triangular shape with the base upward. There is a dorso-cervical kyphosis, the clavicles are prominent and the legs are bent forward

and outward. The stature of the patient becomes shortened. The cause of the disease is unknown.

No treatment seems to be of any service.

IX. OSTEOMALACIA—MOLLITIES OSSIUM.

Etiology.—A rare disease of early adult life. It is common in women, particularly in pregnant women.

Morbid Anatomy.—The bones are extensively decalcified from the medullary canal outwards, so that they are easily bent. The pelvic bones become pushed inwards, so that the transverse diameter of the pelvis becomes shortened. The promontory of the sacrum is pushed forward. The rami of the pubes may run nearly parallel to one another. The long bones are bent.

Symptoms.—The stature of the patient is diminished, the legs much bent, the arms less so, the pelvis compressed. The spinal column and the ribs in time become deformed. Lime salts may be excreted in the urine. The disease proves fatal in time.

X. ACHONDROPLASIA.

This is a dystrophy of the epiphyseal cartilages. They are invaded by connective tissue from the periosteum, which causes premature union of epiphysis and diaphysis and prevents growth in length. The extremities are thus very short, the fingers reaching about the crest of the ilium when the arms are placed at the sides, and while this deficiency in length is due to all the long bones, the humerus and femur are more at fault than the arm and leg bones. The root of the nose is depressed, but the head and trunk are

about the usual size. The hand is short and has a trident shape. The signs of rickets are absent.

DISEASES OF NERVES.

NEURALGIA.

Neuralgia is nerve pain, and nerve pain may have a definite organic basis, or may be purely functional. The organic group of causes may be further subdivided into those in which either (1) a toxin or other irritant acts locally on some portion of the sensory neurone, or (2) a tumor presses on or otherwise involves a nerve, or (3) the sensory neurones are badly nourished with blood and as a result are more liable to be stimulated by reflex irritation, such as may be induced by a carious tooth, digestive error and similar conditions.

The functional group of neuralgias are somewhat difficult to differentiate from this third subdivision, but there are certainly cases in which pain occurs in a sensory nerve or nerves without any organic cause whatsoever being present, and therefore it is necessary and advisable to retain this functional group, although remembering its close association with the third subdivision just referred to.

Pathological Anatomy.—In cases of severe neuralgia there is usually swelling over the region of the nerve, and the skin belonging to the area may be puffy and œdematous. There is often some inflammatory effusion into the nerve itself, and in certain cases there may be a definite change in the nerve-fibres. Where the nerve suffers seriously from the presence of an effusion, there will inevitably be changes in the central nerve cells of the affected neurones. Some authorities would prefer to include these cases under the category of neuritis, but they are nevertheless true cases of nerve pain, and therefore must find a place here.

Symptoms.—Pain varies with the type of neuralgia present, from a dull ache to the sharpest and most agonising paroxysm. There are generally painful points which correspond to the position of foramina or grooves in the bone through or along which the affected nerves pass, and, as already mentioned, the skin supplied by the nerve or nerves in question may be swollen and is often hyperæsthetic. The hair in the area of the affected nerve may become white, and local sweating is not uncommon. The pain may last for minutes or hours, and there is often a definite periodicity, for example, trigeminal neuralgia may come on every night when the unfortunate patient retires to bed.

There are many varieties of neuralgia, and a brief description of the more important of these is requisite.

(1) *Trigeminal neuralgia, neuralgia of the fifth nerve or tic douloureux*.—Any of the three branches of the fifth nerve may be involved. When the *first* or *ophthalmic division* suffers, the pain is localised to the region of the supraorbital notch, and pressure over that point, just above the upper eyelid, is intensely painful. The eyeball is tender to the touch, and sometimes associated with this type of neuralgia there is conjunctivitis, and, more commonly still, iritis. As a rule, actual eye inflammation is associated with a herpes frontalis. Where the *second* or *superior maxillary division* of the nerve is involved, in the majority of cases the cause of the neuralgia is to be found in one or more of the upper teeth on the affected side, and pain is generally severe, just at the infraorbital foramen, where the nerve appears on the cheek. Care should be taken in a persistent case of neuralgia of this type to investigate the condition of the antrum, because empyema of that cavity may account for prolonged pain. Where the *third* or *inferior maxillary division* of the nerve is involved, the cause is usually to be found in one of the lower teeth on the affected side, and the painful point may be either at the mental foramen or where the auriculo-temporal branch of the nerve

passes up behind the ear. Chewing is peculiarly painful where a dental cause of pain is present, and sometimes twitchings of facial muscles are associated. Most so-called paroxysmal neuralgias are affections of the fifth nerve. The pain in such a case is excessively severe, is apt to recur on the slightest provocation, and may even threaten the mental equilibrium of the sufferer.

(2) *Neuralgia of the spinal nerves* is by no means uncommon. Tumors, injuries, fractures, syphilitic lesions and possibly exposure to cold may all act as definite causes of pain, limited to one or other of the intercostal or abdominal nerves.

Neuralgia of the breast or mastodynia is a not infrequent affection in cases of over-lactation, or less frequently at the menstrual epoch in young girls.

Neuralgia of the brachial plexus may result from injury to the neck or shoulder-girdle, or may be associated with rheumatism, gout or other allied conditions. There are also cases of trade spasms which are in part a neuralgia of nerves supplying the muscles chiefly affected. The commonest examples of such trade spasm are to be met with in clerks, pianists, telegraphists and typists.

Intercostal neuralgia is commonly associated with herpes zoster, and the three painful points correspond to the distribution of the cutaneous branches of the affected intercostal nerve. There is reason to believe that in herpes zoster the irritant rather involves the posterior nerve root ganglion than the nerve itself after its formation by the junction of the two nerve roots. The pain in these cases varies considerably in intensity, but is apt to be long-standing and of considerable severity. *Lumbo-sacral neuralgia* is very similar to the last, only at a lower level than the intercostal nerves, and its development may be associated with the presence of a pelvic tumor, with constipation, and, in addition, in some cases, with a predisposing cause of ill-health, such as an attack of influenza.

Sciatica is one of the most important forms of neuralgia of a spinal nerve. It is often due to an interstitial neuritis or to a perineuritis, and another common cause of pain is pressure on the nerve or nerve roots in the pelvis by tumor, a loaded rectum or other definite condition. It is therefore rarely a functional neuralgia. The majority of cases of sciatica are due to exposure to cold in persons subject to rheumatism. Pressure in the pelvis is the next most common cause, and in every case of persistent sciatica a careful examination should be made so as to eliminate the possibility of the pain being of pelvic origin. The nerve is swollen in the rheumatic cases and there is evidence of an inflammatory effusion. These effusions may be between the nerve bundles or inside the funiculi, and in certain cases may be hæmorrhagic. It is the presence of these effusions which suggests that acupuncture is a good method of treatment.

The pain in sciatica often comes on gradually, is more severe at night, and shoots down the leg to the knee or to the heel. It is accentuated by any position which increases the pressure of the muscles on the affected nerve. Stooping with the leg straight is impossible, and the patient finds that he cannot sit comfortably on a hard seat. He may be forced to walk with the leg bent, and he may complain of certain points as being especially tender. The chief tender points are above the hip joint, in the popliteal space, just above and behind the head of the fibula and immediately behind the external malleolus. Where the nerve-fibres are damaged the muscles may eventually waste, while cramps or spasms of the affected muscles are not uncommon in the earlier stages. Relapses are apt to occur where the affection is of rheumatic origin.

In diagnosing cases of sciatica, care should be taken to eliminate the possibility of the pain being due to synovitis or disease of the hip joint, and in every case pressure should be applied along the line of the sciatic nerve so as to discover whether it is really tender to the

touch, and the pelvis should be carefully explored for any tumor or other cause. The prognosis depends much on the duration of the affection and the amount of muscular atrophy present. There is no disease which demands such careful and painstaking treatment, and which if neglected is apt to lead to such dire results.

Neuralgias are not uncommon in connection with the genital organs, and especially the testicles or ovaries.

Coccygeal neuralgia is apt to follow labour and any condition in which pressure on the coccyx, as in the course of parturition, has given rise to definite damage; but it is also a common affection in neurasthenic and hysterical patients, and in such cases may give rise to much pain and distress to the sufferer.

Metatarsal neuralgia or Morton's affection of the foot is one of the most important because one of the commonest forms of neuralgia in connection with the foot. It consists in severe attacks of pain localised to the base of one or other of the metatarsal bones, and in particular those of the second and fourth toes. The pain shoots up the leg and is increased by pressure over the affected metatarsal. It is a result of wearing shoes which are too tight, and especially where the patient is flat-footed. Sometimes excision of the head of the metatarsal is called for, but, generally speaking, raising the instep and inducing the sufferer to wear wider shoes are sufficient to relieve the pain.

Neuralgias of *visceral origin* are not uncommon, and the crises of locomotor ataxia are instances of this type of neuralgia. They may occur in the stomach, œsophagus and elsewhere. *Pelvic neuralgia* is also common, especially in nervous and hysterical women, although it should be remembered that its presence may indicate some organic lesion of importance.

Treatment of neuralgia.—The principles which govern the treatment of neuralgia are threefold. First, determine,

if possible, any local or exciting cause for the pain. Second, investigate the possibility of a predisposing factor in the case, whether a constitutional element or the existence of a lowered state of the general health. Third, administer analgesics, such as those belonging to the coal-tar group of remedies, for example, antipyrine, phenacetin, and so forth. Associated with this third plan of attack may also be mentioned the application of local analgesics and various surgical operations such as nerve stretching, excision of portions of the nerve, or even a complete division of the nerve or nerves responsible for the pain.

In cases of *trigeminal neuralgia* any constitutional factor present, such as rheumatism, gout and malaria, should be appropriately treated and a careful investigation made of the eye, especially with regard to sight, of the upper and lower teeth, the nose and the antrum. The branch of the fifth nerve affected indicates where the exciting source of irritation should be sought for. Any digestive disturbances must also be corrected. The analgesic remedies commonly ordered include quinine in relatively large doses (5 to 15 grains); phenacetin (20 grains); antipyrine (10 to 20 grains); and ammonol (2 to 5 grains). Where pain is associated with the teeth, quinine and antifebrin will be found of special value. Butyl-chloral hydras (10 to 20 grains) and exalgin (2 to 3 grains) have attained a considerable notoriety for their prompt action in the relief of trigeminal pain whatever the cause may be. Certainly, remedies such as aconite and tincture of gelsemium are of signal value where the patient is strong, but over-depression should be carefully avoided, and the administration of most of the remedies just referred to, and especially those which have the most prompt effect, often demand the use of a diffusible stimulant, such as alcohol, taken with or soon after the administration of the drug. The baneful effects of alcoholism and the dangers which may follow the morphia habit are too well known to make it necessary to do

more than to remind the reader of their importance. In fact, although the value of alcohol and opium in the treatment of certain cases of trifacial neuralgia cannot be denied, neither should be given unless absolutely necessary, and only by the express orders of the physician.

Local applications such as menthol, a mixture of chloral and camphor in equal parts, and ointments such as aconitine, may be applied to the skin over the painful nerve. Osmic acid (2 to 3 minims of a 1 per cent. solution), weak solutions of cocaine, or 80 per cent. alcohol may be injected into the affected branch of the nerve. Local massage and sometimes the application of galvanism, especially with the positive pole, have been found of advantage in certain cases. The surgical procedure of section of the branch of the nerve or the more radical measure of extirpation of the Gasserian ganglion has been successfully carried out where the neuralgia is paroxysmal and has resisted all other methods of treatment.

Neuralgias of the breast are often relieved by the application of a local sedative such as extract of belladonna (the alcoholic extract with equal parts of glycerine), while in certain brachial neuralgias local counter-irritation, in other cases local sedatives, afford relief. The neuralgia forming a part of any trade spasm is generally benefited by stopping the movements to which the affection is due.

Appropriate treatment for *herpes zoster* is described on page 939.

Sciatica should in most instances be treated with salicylates and other anti-rheumatic remedies, although in every case a possible pelvic cause for the condition should be sought for, and where found removed if practicable. As effusions are nearly always present in cases of sciatica not due to pressure, rest should be enjoined, sometimes simply by confinement to bed, in other cases by the use of the long splint. There is a stage in many cases of sciatica in

which local poulticing or more vigorous counter-irritation by blistering are advantageous, and, in the writers' experience, when the pain continues notwithstanding the employment of these measures, acupuncture should not be delayed. In carrying out this procedure, the line of the nerve should first be marked out on the back of the thigh. Half a dozen aseptic sciatica needles should be inserted into the nerve, care being taken that the highest needle should be *below* the line of the fold of the buttock, and the lowest needle well *above* the popliteal space. The needles should be left in position for about half an hour, the patient lying on his face with the thigh covered with a cage so as to keep off the pressure of the bed-clothes. The object of the treatment is to allow some of the exudation to escape, and dexterously performed the patient is only aware of the insertion of two or at most three of the needles. Injections of osmic acid or cocaine into the nerve, although they have been recommended, are more likely to do harm than acupuncture. Where sciatica does not yield rapidly to treatment, and in cases in which it has become chronic before the patient seeks help, the use of hot baths, and especially mud baths, may be found of advantage, but probably these cases may require nerve stretching, or the removal of a portion of the nerve should the pain continue very severe. Nerve stretching is easily performed, the amount of strain exercised on the nerve being that which is found sufficient to lift the leg off the table, and it is supposed that adhesions, to which the pain may largely be due, are broken down by this procedure. Where the muscles have wasted, massage and other measures should be adopted for keeping up nutrition, but no such treatment should be commenced until the inflammatory stage of exudation is over.

An indication has already been given of the method of treatment for *metatarsal neuralgia*, and it is only requisite to add here that in all cases where neuralgic pain is associ-

ated with nerves situated in the neighbourhood of the pelvis, tumors or anything causing pressure should be searched for, and where found removed if possible.

Coccygeal neuralgia may demand the excision of the coccyx or the division of adhesions, but as many of these cases are more functional than organic, nerve tonics and anti-hysterical treatment will frequently be found of the greatest value.

NEURITIS.

This may be local or general. Local neuritis implies limitation to one nerve or part of a nerve or limitation at all events to nerves situated in close proximity to each other. General neuritis, commonly called multiple or peripheral neuritis, involves many nerves, although, as the name implies, the extremities of the body are most affected.

LOCAL NEURITIS.

Inflammation may specially involve the sheath of a nerve when the condition is called *perineuritis*, or it may affect the connective tissue between the nerve-fibres in the funiculi. Whether the inflammation is limited to the connective tissue inside or outside the funiculi, the term *interstitial* neuritis is applied to it. Inflammatory or degenerative changes may involve the nerve-fibres themselves, and to this the term *parenchymatous* neuritis is given. Local neuritis, while generally of interstitial type, may also be primarily, or more commonly secondarily, parenchymatous. It is easy to understand that where there is an involvement of the interstitial tissue the nerve-fibres must suffer secondarily, and the opposite also holds good.

Etiology.—A localised neuritis is commonly the result of exposure to cold, while wounds and bruises and other

injuries of the nerve, including injections of various drugs for the relief of pain or other purposes, may all cause the condition. Rheumatic subjects are specially liable to suffer from the neuritis resulting from exposure to cold, and it seems probable that in alcoholic neuritis, which is primarily parenchymatous, exposure to cold may determine the paralysis of a special limb or limbs. Inflammation from a neighbouring focus may also involve a nerve, and it is desirable to include under this heading trade paralyses dependent on the too frequent use of certain nerves with their groups of muscles, and in which there may be, although very exceptionally, an inflammatory lesion.

Pathological Anatomy.—In inflammatory cases there is generally an exudation of lymph, possibly also of leucocytes, into the affected nerve. The nerve-fibres themselves may degenerate and resemble what is described under multiple neuritis.

Symptoms.—Pain is the chief clinical feature, and later there may be evidence of paralysis. The pain shoots down the line of the nerve, is increased on pressure, and is often more severe at night time. The skin, at first hyperæsthetic, may eventually become anæsthetic. Paralysis of muscles may develop if the nerve carries motor fibres, and we may find tenderness on gripping the muscles and sometimes fibrillary twitchings. Trophic changes are not uncommon and consist in reddening or glossiness of the skin, brittleness of the nails, local sweating, and sometimes eruptions. A neuritis at first limited to a part of a nerve may spread upwards, causing the ascending neuritis referred to later.

Diagnosis.—As a rule there is little difficulty from the very typical symptoms which most cases present.

Prognosis.—This depends largely on the severity of the lesion and of the possibility of removing any etiological factor which may be keeping up the inflammation.

Treatment.—Rest to the affected part should be insisted on, together with the application of warm fomentations or poultices, while any rheumatic, gouty or syphilitic element in the case should be appropriately treated. Sometimes more vigorous counter-irritation is called for, such as fly blisters or iodine, and it is certainly prudent to consider whether puncturing the nerve might not be beneficial. Galvanism is sometimes of use, not merely for the relief of pain but also for the removal of exudation.

ASCENDING NEURITIS—NEURITIS MIGRANS.

In *ascending neuritis*, the inflammation ascends, causing intense pain, often so severe as to determine the patient in favour of having the affected limb amputated. It is the result of a septic wound, or at all events indicates the presence of an irritant which causes the ascending neuritis.

The term *migrans* has been given to a neuritis which migrates or flits about from one part of a nerve to another. The cause should be removed where this is discoverable, and in the first instance nothing further may require to be done. If ineffectual, nerve section is called for, or even amputation.

SYMPATHETIC NEURITIS.

It sometimes happens that as the result of the inflammation of one nerve the corresponding nerve on the opposite

side of the body may be sympathetically affected. The lesion varies in nature.

MULTIPLE NEURITIS—PERIPHERAL NEURITIS.

This is a group of slightly different forms of neuritis. The lesion specially involves the peripheral parts of certain nerves, although it seems probable that the neurones suffer together. Nearly all forms of this neuritis are essentially of parenchymatous type.

Etiology.—The following grouping of causes* will be found helpful, although each group may not be absolutely distinct from its fellows:—(1) Diffusible stimulants as alcohol, naphtha, bi-sulphide of carbon, dinitro-benzine; (2) metallic poisons and specially arsenic, lead and mercury; (3) toxins from the micro-organisms of diphtheria, typhoid, rheumatism, septicæmia, syphilis, pneumonia, tubercle, malaria, beri-beri, leprosy, &c.; (4) it may also occur in diabetes mellitus, possibly in gout, in cachexia, cancer, and sometimes in profound anæmia. There is no question that these etiological factors act more potently where the individual is exposed to cold, or is suffering from the effects of some severe illness or from profound mental depression. It should be noted that a number of the forms of neuritis included under this etiology are primarily interstitial rather than parenchymatous in nature.

Pathological Anatomy.—The nerve-fibres show typical degeneration, the axis-cylinders have become broken up, the myelin sheaths are represented only by a collection of droplets, and the neurilemma nuclei have proliferated. In

* *A Short Practice of Medicine* by R. A. Fleming, page 545.

our opinion there is almost always a marked inflammatory exudation into the affected funiculi, sometimes hæmorrhagic and sometimes consisting largely of leucocytes, although more generally it consists of inflammatory lymph. The muscles affected show marked degenerative changes, mostly in the direction of loss of striation, fatty degeneration and proliferation of nuclei.

Clinical Features.—It is best to describe first a case of alcoholic origin as a type of multiple neuritis. It is common in women, it occurs in persons of all classes and at all ages, but it is certainly more frequently met with in those in whom bad health aids the work of the toxin. It is difficult to say which form of alcohol is most apt to cause the disease.

There are several distinct types of alcoholic neuritis which depend on whether motor or sensory fibres have specially suffered, and this selective peculiarity of the toxin for special neurones is by no means uncommon.

The *sensory* changes generally present include pain, with tingling and numbness; the pain is increased by pressure over the affected nerves and may be caused by squeezing the muscle with the hand, while cramps are by no means uncommon. There is a remarkable delay in the conduction of sensory stimuli, the individual not crying out until some seconds after pressure has been applied to the nerve or muscle. There is also, not infrequently, some confusion or blunting of temperature sense impressions.

The *motor* manifestations include wrist-drop and foot-drop due to paralysis of the extensors of the hand and foot. There is high-steppage gait and weakness of many of the other muscles of the body, amounting to paralysis. There is often tremor, or what is better described as tremulousness, of the limbs. In the legs the extensor muscles, the peronei, and the tibialis anticus are specially apt to be paralysed, and later the intercostal muscles and the diaphragm may

become affected. Inco-ordination is a marked feature in certain cases. The deep or tendon and the superficial *reflexes* are usually lost, although cases occur in which the knee-jerks may be exaggerated, at all events to begin with. It is rare to find bladder or bowels interfered with. The *electrical reactions* are—diminished irritability to faradism and very frequently the definite development of the reaction of degeneration (see page 839 for full description). The *vaso-motor phenomena* include pallor, redness and swelling of feet and hands, and a peculiarly malodorous perspiration. The *trophic changes* include wasting of muscles, glossiness of the skin, brittleness of the nails, and not infrequently the appearance of bed-sores. The patient's mental condition is remarkable. There is great loss of memory, associated with the most striking and vivid hallucinations. The patient describes a visit which he says he has paid that morning, generally in quest of liquor, although he may be absolutely unable to get out of bed.

It must not be supposed that all these clinical features will be equally distinct in every case, because in some sensory and in others motor symptoms predominate. There is a distinct tendency, towards the termination of a case, to the involvement of the vagi nerves as well as the intercostals and the phrenics, and death is not uncommonly due either to heart failure, a low type of pneumonia, or sheer debility.

In *arsenical neuritis* or the neuritis due to mercury, the clinical description is not very dissimilar excepting that there is more pronounced inco-ordination and tremor as a general rule in mercurial cases, while in those due to arsenic, the pigmentation of skin and a keratosis of the feet and hands are distinctive.

In *lead poisoning*, paralysis, closely resembling the alcoholic type in nature, may be met with, but it is often more limited, giving rise to typical wrist-drop, al the supinator longus and extensors of the thu :

In a second type of lead paralysis the upper arm muscles may be paralysed, including the deltoid, biceps, brachialis anticus, and supinator longus, although the pectoral muscles escape. In a third type the small muscles of the hand are affected in a manner similar to what is seen in progressive muscular atrophy. In a fourth type it is the peronei, the extensor longus digitorum and the extensors of the great toe which are involved, causing a distinctive foot-drop. The fifth and last type is a laryngeal one, and in it the adductors of the glottis are paralysed.

Diagnosis.—The paralysis is peripheral and is, as a rule, symmetrical, while the pain felt on pressure over affected nerves and muscles is almost distinctive. In infantile paralysis, Landry's paralysis, and in hysteria these sensory manifestations are not present as a general rule. It must not, however, be forgotten that the motor type of multiple neuritis is very closely allied to Landry's paralysis.

Prognosis.—Acute cases often prove fatal, and generally terminate by the implication of the respiratory nerves and muscles. In no acute case is it advisable to offer too favourable a prognosis. Chronic cases of alcoholic neuritis and limited paralysis, the result of lead poisoning, are likely to recover.

Treatment.—Promptly remove the cause of the condition. Endeavour to eliminate lead or other metallic poisons from the system by the use of potassium iodide and saline purgatives. In alcoholic cases absolute rest should be prescribed for the inflamed nerves, and either hot fomentations or counter-irritation should be tried. In certain instances acupuncture should be thought of, although its usefulness is limited to those cases in which an exudation is present into the funiculi. Care should be taken to correct any tendency to contracture in long-stand-

ing cases, and for foot-drop the use of sand-bags is often of value. Once the inflammatory stage is over, hypodermics of strychnine may be given into the affected limb, and galvanism and faradism are both of value in keeping up the nutrition of the muscles. Faradism is naturally restricted in its use to cases in which the nerve fibres can still conduct impulses or have begun to regain their former conductivity. Internally, and especially in alcoholic cases, mental excitement may require the use of bromides, chloral, hyoscine or other powerful sedative.

It is difficult in alcoholic cases to insure the patient's sobriety in future.

TUMORS OF NERVES.

There are a number of different kinds of tumors which demand a few words of description. Some of these are *true neuromata*, by which is implied that they consist of actual nerve tissue, either fibres or cells. The majority are *false neuromata*, in which the tumor consists largely of connective tissue.

1. *Plexiform neuromata* are invariably multiple and consist of tortuous bundles of nerve-fibres with a considerable amount of myxomatous tissue. They may occur in any of the nerves of the body, although not uncommonly they are found in connection with the fifth nerve. They are of embryonic origin and are often associated with a definite hereditary history.

2. *Multiple neuro-fibromata* or von Recklinghausen's disease is a remarkable form of nerve tumors presenting the following characteristics:—(1) There are many small fibrous tumors situated on the nerve trunks, and in connection with these there are numerous fibrous nodules, varying in size, situated in the skin. These nerve tumors

cause pain, often very severe muscular cramps, and sometimes even paralysis; (2) the skin is pigmented in patches varying in size and distribution; (3) there is, lastly, a remarkable mental degradation and loss of memory, with difficulty in speaking, which sooner or later manifests itself in persons suffering from this remarkable disease.

3. *Multiple neuromata*, also called *tubercula dolorosa*, sometimes involve the terminal twigs of peripheral nerves and they form, as the name would imply, extremely sensitive tumors, found specially on the face, breast or in connection with the joints.

4. *Amputation neuromata* are an inevitable sequel to section of a nerve where there is no chance of reunion. The small tumors are formed largely of growing axis-cylinder processes with their medullary sheaths. They are only likely to cause trouble when, owing to the position of the tumor, it is pressed upon in the stump, as, for example, when walking. These amputation neuromata if painful may require excision, but they are usually prevented from giving trouble owing to the care which surgeons take to cut through the nerves as high up as possible in the course of an amputation. *Carcinomatous* and *sarcomatous* nerve tumors do not require any description.

DISEASES OF SPINAL NERVES.

I. PARALYSIS OF THE PHRENIC NERVE.

This nerve arises from the fourth and to a less extent from the third or fifth cervical nerves. It may be injured by fractures, wounds or tumors in some part of its course, and in cases of peripheral neuritis, and especially in that due to

diphtheria or alcoholism, parenchymatous changes may occur.

Symptoms.—In most cases paralysis of the diaphragm is bilateral, and as a result breathing is restricted to the thoracic muscles. During inspiration, in place of the abdomen being distended, it is retracted and the slightest exertion causes intense dyspnœa. The lungs are apt to suffer, bronchial secretion being retained, and septic pneumonia induced at the bases. Coughing, largely due to the contraction of the diaphragm, is rendered difficult or impossible. Where the lesion is unilateral, a careful examination of the upper part of the abdomen demonstrates the absence of movement on the paralysed side during inspiration.

Diagnosis and Prognosis.—The diagnosis is easy, and the prognosis depends on the nature of the condition. It should be remembered that paralysis of the diaphragm in diphtheritic or alcoholic neuritis is often one of the terminal phenomena indicating the approach of a fatal result. In bilateral cases of paralysis of the diaphragm artificial respiration should be kept up, and it is certainly desirable to try the effect of oxygen inhalations.

SPASM OF THE DIAPHRAGM OR HICCUGH.

Hiccough or hiccup is the result of spasmodic contraction of the diaphragm. It is reflex in its production, and is generally caused by irritation in the stomach or abdomen, in fact it is due to irritation of some peripheral branch of the vagus nerve. There are also cases where the irritation is central or where a toxin acts on one or more centres, probably in the medulla. It is well to remember the frequent occurrence of hic-

cough in cases of uræmia, delirium tremens and in some other cases of profound toxæmia.

Treatment.—Common remedies for hiccough, often efficacious in trifling cases, are the following:—sipping a considerable quantity of cold water without drawing breath, what is aimed at being repeated acts of swallowing and not so much the quantity of water swallowed. Another method is endeavouring to drink water from the opposite side of the tumbler, the effort requisite to perform this feat being apparently sufficient to arrest the diaphragmatic contractions. In severe cases an ice-bag may be applied over the stomach and ice administered by the mouth, or such sedatives as morphia, dilute hydrocyanic acid (3 minims) or bromides may be given. Hypodermic injections of hyoscine are worth a trial when all else has failed. Washing out the stomach, even if that organ does not contain any irritating ingesta, has been found beneficial in a considerable number of cases.

II. PARALYSIS OF THE BRACHIAL PLEXUS.

Blows or wounds to the side of the neck or the shoulder girdle may damage the whole or part of the brachial plexus, and severe fractures of the clavicle are not infrequently accompanied by some injury to the brachial plexus. Where the arm is forcibly dragged, the lower spinal nerve roots forming the plexus may be torn through. Such cases have occurred in factories, and sometimes from traction of the arm during parturition, the fulcrum being, as a rule, the lower edges of the ribs over which the nerves are tightly stretched. There are two types of arm paralysis due to lesions of the brachial plexus which demand separate description. A cervical rib may also cause pressure paralysis.

*A. UPPER ARM TYPE OF PARALYSIS—ERB'S
PARALYSIS.*

This is a lesion of the fifth and sixth cervical roots. It is generally the result of injuries and blows to the side of the neck, and it has occurred where heavy loads have been carried on the shoulder.

Symptoms.—The deltoid, biceps, brachialis anticus, supinator longus, and sometimes the supinator brevis and the supra- and infra-spinatus muscles may all be paralysed. As a result the arm cannot be abducted or flexed at the elbow-joint, and supination is impossible. The anæsthesia is limited to the outer side of the upper and lower arm. Pain may be present in some cases.

*B. LOWER ARM TYPE OF PARALYSIS—KLUMPKE'S
PARALYSIS.*

This is a lesion of the eighth cervical and first dorsal roots. It is this form which generally results from traction upon the arm, although it may also be produced by tumors, meningitis and so forth.

Symptoms.—The small muscles of the hand and the flexors of the fingers and the forearm are paralysed, and if the lesion also involves the sixth and seventh cervical roots, an even wider range of muscles may be paralysed. The muscles affected waste. There is loss of sensation in the realm of the ulnar nerve, and there is evidence of the involvement of the first dorsal root by the changes in the eye on the affected side. These changes include contraction of the pupil, a sluggish pupil reaction and diminution in size of the palpebral aperture, with sinking in of the eyeball and a slight consequent ptosis. These phenomena

depend on a lesion of the connections of the first dorsal root with the sympathetic.

C. SERRATUS PARALYSIS.

The long thoracic nerve may be injured by pressure or by a wound, and is sometimes affected by neuritis.

Symptoms.—The muscle fixes the scapula when the arm is elevated above the horizontal position. When the arm is raised vertically, the scapula being fixed by the rhomboids and other muscles, the serratus can elevate the ribs, and so help to expand the chest. Where the serratus is paralysed, these movements are impossible, and the marked projection of the scapula in certain movements renders the diagnosis extremely easy. The trapezius muscle is not infrequently also affected.

D. PARALYSIS OF THE CIRCUMFLEX NERVE.

This nerve arises from the posterior cord of the brachial plexus. It supplies the deltoid and teres minor muscles. It is one of the forms of crutch palsy. Owing to the deltoid paralysis the arm cannot be raised. The paralysed muscles waste.

E. PARALYSIS OF THE MUSCULO-SPIRAL NERVE.

This nerve is often injured by the pressure of a crutch, or where an individual falls asleep with the arm over the back of a chair. Fractures and other injuries may also cause musculo-spiral paralysis.

Symptoms.—In most cases where the lesion is high up all the muscles supplied by the nerve are paralysed, and the paralysis of the triceps, the supinators and the extensors of the wrist, fingers and thumb is specially marked. The

elbow cannot be extended, the hand is pronated and wrist-drop occurs. The area of anæsthesia is limited to the outer part of the forearm and the back of the thumb, index, middle, and half of the third fingers, but the terminal phalanges escape.

F. PARALYSIS OF THE ULNAR NERVE.

The ulnar nerve arises from the last cervical and first dorsal nerves. Injuries to the elbow or shoulder may be responsible for the paralysis.

Symptoms.—The flexor carpi ulnaris, the flexor profundus digitorum (ulnar half), the interossei, the two ulnar lubricales, the hypothenar muscles, the adductor pollicis and the deep head of the flexor brevis pollicis are paralysed, causing the typical claw-like hand. There is over-extension of the metacarpo-phalangeal joints, and flexion of the inter-phalangeal. The sensory changes include an area of anæsthesia extending to the wrist, and limited to the ulnar side of the hand (the little finger and ulnar half of the third finger).

G. PARALYSIS OF THE MEDIAN NERVE.

The median nerve arises from the lower four cervical roots. The nerve may be injured by a lesion in the forearm.

Symptoms.—The flexors of the fingers (excepting the ulnar half of the deep flexor), the flexors of the wrist (excepting the flexor carpi ulnaris), the two pronators, several of the thumb muscles and the radial lumbricales are paralysed. The paralysis prevents pronation, the wrist cannot be flexed towards the radial side, and the thumb cannot be brought into opposition to the tips of the fingers. The sensory changes are limited to anæ-

thesia of the radial side of the hand, including the palmar aspect of the thumb, and the first, second and half of the third fingers, and also the back of the same fingers and the last phalanx of the thumb.

III. THE LUMBAR PLEXUS.

This arises from the first three lumbar roots, together with half of the fourth, and has a connecting branch from the last dorsal root. It innervates the flexor and adductor muscles of the thigh, the extensors of the knee, and the cremaster muscle. The sensory supply includes the skin over the lower part of the abdomen, the region of the genital organs, the greater portion of the thigh, and the inner surface of the leg and foot. Tumors, vertebral caries and inflammation of the meninges are not uncommon causes of involvement of the plexus, in whole or in part, while sometimes a parenchymatous neuritis occurs.

The *obturator nerve* may be pressed upon by tumors in the pelvis, by pressure during labour, or by an obturator hernia. The nerve supplies the adductors of the thigh, and the external rotators, and when it is paralysed the legs cannot be crossed. The *anterior crural nerve*, which is also a branch of this plexus, may be damaged by a lesion of the hip joint, by abscesses or tumors. The nerve supplies the extensors of the knee, and sensation to the greater part of the thigh and inner side of the leg, below the knee. The *external cutaneous nerve* is not infrequently affected by neuritis, and gives rise to pain, especially felt in the region of Poupart's ligament.

IV. THE SACRAL PLEXUS.

The sacral plexus arises from the first three and part of the fourth sacral nerves, and supplies the extensors and

rotators of the thigh, the flexors of the knee, and the foot muscles. The sensory supply includes the skin over the buttock, the back of the thigh, the back and outer side of the leg below the knee, and most of the skin of the foot. Paralysis of the plexus may be the result of tumor in the pelvis, or injury during labour.

The *sciatic nerve*, which is the chief branch of this plexus, may be injured by pelvic lesions such as tumors, and fracture of the femur may also involve the nerve. Neuritis is not uncommon, due to one or other of the causes above indicated, or it may be the result of rheumatism and so forth. The nerve supplies the extensors of the hip, the flexors of the leg, and the muscles below the knee. Wasting of the paralysed muscles, and sometimes trophic inflammation of the skin, follows a lesion of the nerve. The external and internal popliteal branches of the sciatic may suffer separately from injury or from neuritis. The former supplies the extensors of the toes, the peronei muscles and the tibialis anticus, and the skin of the outer half of the front of the leg below the knee and the back of the foot. Typical foot-drop, with high steppage gait, follows a paralysis of this nerve. The internal popliteal nerve supplies the tibialis posticus muscle, the muscles of the calf and the sole of the foot, the popliteus and long flexors of the toes, and also part of the skin of the outer side of the leg below the knee and the outer edge and sole of the foot. Paralysis of this nerve renders the patient unable to extend the foot at the ankle joint, to abduct the foot, and to stand on tiptoe.

Treatment.—Treatment of the above conditions depends on the nature of the lesion in each case. Where there is a tumor, or where the nerve has been divided, surgical interference may be necessary. In cases of neuritis, rest and, when inflammation has subsided, massage

and electrical treatment are indicated, so as to keep up the nutrition of the affected muscles.

DISEASES OF CRANIAL NERVES.

FIRST OR OLFACTORY NERVE.

The sense of smell plays an important part in gastro-nomic pleasures, and in testing the sense of taste the part played by the functions of the first nerve must be kept in mind. By means of the taste fibres alone we can recognise only four varieties of taste, namely, sweetness, saltiness, bitterness and acidity, and the olfactory nerve supplies the rest of what we usually ascribe to our sense of taste.

Anosmia.—Loss of smell may be due to a lesion of the cortical olfactory centre in the brain, of the olfactory bulbs and tracts, or of the nasal mucous membrane. The last is the commonest site, and the most familiar instance is an ordinary cold in the head. *Ozæna* and other organic diseases of the nasal passages may also be responsible. It must be remembered that a lesion of the fifth nerve may cause dryness of the nasal mucous membrane, and so interfere with the perception of smell.

Falls or blows on the head, and rarely tumors and other intracranial lesions, may damage the olfactory bulbs or the nerve fibres which pass from them through the cribriform plate of the ethmoid bone. In locomotor ataxia primary degeneration of the olfactory tracts and bulbs sometimes occurs. Occasionally one olfactory bulb is congenitally absent, and in such cases both sides of the nose may be supplied from one bulb alone. The cortical centre for smell, situated probably at the tip of the uncinate convolution, is sometimes, though rarely, the site of lesion.

Smell may be tested by asking the patient to sniff up oil of peppermint, clove, asafoetida, &c., but in no case should the agent used be one which, like ammonia, will also stimulate the terminations of the fifth nerve in the nasal mucosa.

Hyperosmia and Parosmia are generally insane delusions. The former implies excessive sensitiveness to smell, and the only important instance of this from a purely medical standpoint is where the aura of epilepsy is one of smell, and a smell which may be peculiarly overpowering, but which might with greater accuracy be termed an instance of parosmia. Parosmia is a hallucination of the sense of smell, and its interest is largely limited to alienists.

THE SECOND OR OPTIC NERVE.

The reader is strongly urged to revise his anatomical knowledge of the optic paths. It is only possible to offer an extremely brief account of these here. The left halves of each retina have their cortical centre in the left occipital lobe, and the right halves in the corresponding region on the right side. The macular part of the retina, or, in other words, the yellow spot, is subserved by cells in the region of the calcarine fissure. The fibres from the nerve-cells in the occipital lobe are gathered together to form the optic radiation of Gratiolet which passes just behind the posterior limb of the internal capsule, and in close proximity to the auditory fibres going to the cochlear nucleus. These optic fibres pass to three intermediate stations or ganglia:—(1) The external geniculate body; (2) the anterior corpus quadrigeminum, and (3) the pulvinar of the optic thalamus. It is important to remember the relationship of the anterior corpus quadrigeminum to the sphincter pupillæ centre in the nucleus of the third nerve. The two are connected by a band of fibres called Meynert's fibres. From these

three ganglia the optic fibres form the optic tract and pass to the chiasma. At the chiasma the fibres divide, the temporal half of each retina being supplied by fibres which do not decussate, whereas the fibres for the nasal half cross in the chiasma. Each optic nerve enters the eyeball slightly to the nasal side of the middle line, and the true posterior pole of the eyeball is the macula, which is therefore situated to the outer side of the optic disc. The macular fibres, which get the name of the maculo-papillar bundle, run together in the optic nerve, and eventually reach the centre of the nerve. The optic disc is the blind spot, its white appearance being largely due to the absence of the vascular choroid which is behind the retina over the remaining part of the fundus. Its edges are sharp and the central artery and vein of the retina come out near the middle of the disc. Owing to the way in which the optic nerve-fibres pass from the nerve through the lamina cribrosa outwards towards the retina, a marked hollowing, commonly called the physiological cup or optic cup, may be noted, and the presence or absence of this cup is of great importance in the diagnosis of optic neuritis and atrophy. The nerve-fibres which are covered by a myelin sheath in the optic nerve lose that sheath when they pierce the lamina cribrosa, so that, as a rule, when they pass into the retina at the edge of the optic disc they are invisible. The optic nerve possesses a pial sheath which is inseparable from the nerve, and from which trabeculae of fibrous tissue penetrate the nerve, dividing up the optic fibres into bundles or funiculi. The arachnoid and dural sheaths are less closely applied, and the former is not a complete membrane, but shows many fenestrations. Effusions can separate these two sheaths from the pial sheath, and occupy the inter-sheath space or vaginal space. In this way great pressure can be exerted at the back of the eyeball on the vessels of the bulb, a pressure which plays an important part in the production of optic neuritis in at all events many cases.

The central artery and vein of the retina enter the optic nerve about 10 mm. behind the eyeball, and soon reach the centre of the nerve, in which position they pass outwards to pierce the optic disc.

The higher cortical sight centres are situated in the angular gyrus and supramarginal convolutions, and the one on the left side of the brain in a right-handed person is the word-seeing centre.

After these preliminary remarks, we shall first describe :—

OPTIC NEURITIS—PAPILLITIS—INFLAMMATION OF THE OPTIC DISC.

Etiology.—It is most common in intra-cranial tumors, in which it occurs in nearly 80 per cent. of all cases. It is practically constant in tumors situated in the cerebellum and corpora quadrigemina, while in neoplasms of the medulla it is, comparatively speaking, rare. It is not a question simply of size of tumor or interference with the circulation of the cerebro-spinal fluid such as by pressure on the veins of Galen which causes the optic neuritis, for a minute tumor in the cerebellum may give rise to the most intense papillitis, while a large tumor, which from its very size must enormously increase the intra-cranial pressure, may cause no optic neuritis whatsoever. Optic neuritis is also commonly met with in certain cases of renal disease, more especially in chronic Bright's disease and cirrhosis of the kidneys. It may result from cysts in the brain, aneurisms, abscess in the brain, and less commonly from meningitis. It may follow profound anæmia, lead poisoning, and sometimes sexual excesses. In many cases it seems probable that a toxin is directly responsible for the inflammation, and the toxic theory of causation of optic neuritis was originally enunciated by Leber.

Pathological Anatomy and Ophthalmoscopic Appearances.—In many cases of optic neuritis due to intra-cranial tumor, the optic disc is enormously swollen, giving the typical choked disc-like appearance, and this is generally associated with greatly increased pressure of lymph in the intersheath space behind the eyeball. Viewed with the ophthalmoscope, the papilla is swollen, the edges blurred, and the colour no longer white but pinkish owing to congestion. The optic cup disappears, the exudation frequently spreads into the retina, and especially along the line of the vessels, and hæmorrhages are common. The arteries are smaller than normal, the veins proportionately greatly engorged and tortuous. Both eyes are usually affected, although it is frequently stated that the eye corresponding to the side of the tumor demonstrates the greater change. It is easy by the direct method of examination with the ophthalmoscope to gauge the degree of swelling of the disc by noting the number of the lens required to render clear the vessels pushed forward by the exudation.

Symptoms.—As a rule sight suffers, but the degree is not always comparable to the intensity of the inflammation of the disc, and sight may remain only partially affected until consecutive atrophy has begun to appear. The fields of vision are limited peripherally, causing what is termed a peripheral scotoma. There is also a marked diminution in the fields for red and especially green. There may be considerable pain, directly due to the optic neuritis, although it may be largely the result of the causal lesion. It is remarkable how rapidly the optic neuritis can disappear after trephining has been carried out. This may be most marked in cases where there is greatly increased intra-cranial pressure. The optic neuritis of renal origin is referred to under the heading of Retinitis.

RETRO-BULBAR NEURITIS.

This is a toxic form of neuritis, the toxin limiting its action to the maculo-papillar bundle and probably the large ganglionic cells in the corresponding portion of the retina. Apparently tobacco and alcohol combined are the best known toxins to which this form of neuritis is due. There is a central scotoma especially for green and red, the fields for which are extremely limited. The disc shows an affection of the temporal side, a segment of the papilla being first swollen and blurred, and later white and atrophied. The toxins believed to be responsible should, so far as possible, be eliminated, and strychnine may be administered along with other nerve tonics.

OPTIC ATROPHY.

I. CONSECUTIVE ATROPHY.

This is the result of optic neuritis. The disc is filled in, or, in other words, the physiological cup is filled in with connective tissue which replaces inflammatory exudation. The disc is usually of a white colour, the arteries are extremely small, and the veins, although to begin with relatively larger in proportion to the arteries, are actually reduced in lumen. White lines, indicating old inflammatory exudation, stretch along the vessels into the retina. Sight is sooner or later absolutely lost.

II. PRIMARY ATROPHY OR GREY ATROPHY.

This is unquestionably the result of the action of a toxin on the nerve-fibres, and possibly also on the ganglionic cells of the retina, from which these fibres pass. It is a not uncommon clinical feature in locomotor ataxia, occurring in 10 per cent. of cases, and it is occasionally found present

in other forms of spinal cord disease. It may also follow the infective fevers, the excessive use of alcohol, lead poisoning and sexual excesses.

The disc is of a grey colour. There are no white lines running along the vessels, and, as a rule, the physiological cup is not filled in to the same extent as in consecutive atrophy. The vessels, both arteries and veins, are equally diminished in size. Sight gradually fails, and the patient generally becomes completely blind.

III. SECONDARY ATROPHY.

This is descending degeneration in the optic nerves, due to pressure on the nerve chiasma or optic tract. The disc is extremely white in colour, the vessels ultimately reduced in size, and the blindness corresponds to the extent of the primary lesion, which may be a hemianopsia, homonymous or heteronymous.

AFFECTIONS OF THE RETINA.

I. RETINITIS.

Retinitis is an inevitable sequel to optic neuritis, although in most cases the retina is only affected in the neighbourhood of the papilla. In *albuminuric retinitis*, the retina suffers far more markedly than the papilla. The region of the macula or yellow spot is chiefly affected, a stellate arrangement of whitish streaks round the macula being perhaps the most typical feature. The disc is often swollen and hæmorrhages are common, both on the disc and also in the layers of the retina. It is supposed that the arterial degeneration so common in chronic Bright's disease and cirrhosis of the kidneys is responsible for fatty changes in the nerve-fibre layers of the retina, and also for the hæmorrhages. It seems probable, however, that a

toxin is responsible for the condition rather than mere deprivation of blood owing to vascular degeneration.

The other forms of retinitis, such as those occurring in syphilis, in leucocythæmia, and in anæmia, demand no detailed description.

II. FUNCTIONAL ALTERATIONS OF VISION.

There are various terms which require brief reference. *Amaurosis* is blindness, and is the term often used in describing the temporary loss of vision following on the use of certain toxic substances, such as alcohol, lead and quinine.

Amblyopia is partial loss of vision, frequently the result of some toxin, and most commonly due to tobacco or alcohol.

Nyctalopia, or night blindness, is the condition in which persons affected see badly in a dim light. The degree varies in different cases, and may be so pronounced as to render the sufferer totally unable to get about once bright daylight is over. The converse of this condition is *Hemeralopia*, in which persons affected find it extremely difficult to see objects clearly in a bright light.

III. AFFECTIONS OF THE OPTIC CHIASMA AND TRACTS.

A lesion of the occipital lobe, of the optic radiation, or of the optic tract causes blindness of the corresponding halves of the retina, and in consequence blindness of the opposite halves of the fields of vision. By means of Wernicke's pupil reaction test it is possible to determine whether the lesion is in front or behind the anterior corpus quadrigeminum, because of the integrity or otherwise of the light-reflex arc. The sensory limb extends up the optic nerve and tract to the anterior corpus quadrigeminum, and from there by Meynert's fibres passes to the sphincter

pupillæ centre in the third nucleus, and the motor limb consists of the fibres passing to the sphincter muscle of the iris. The method of performing the test is to place the patient in a dark room and throw a beam of light on to the affected or blind half of the retina. If the lesion is in the path of the light-reflex arc, no contraction of the pupil results, whereas, if the lesion is behind the anterior corpus quadrigeminum, the pupil will contract normally. The test is by no means an easy one to apply, because it is essential that the ray of light be thrown on the blind half of the retina alone.

Where the lesion is one of the chiasma, the two nasal halves of the retina will probably be affected if it is only the decussating fibres which are damaged. This is one of the forms of heteronymous hemianopsia. Where both temporal halves of the retinae are blind, a double lesion is necessary, one on either side of the optic chiasma, and only involving the fibres of the optic tracts which do not decussate.

Nasal hemianopsia may result from pressure of a small aneurism of one of the anterior cerebral arteries on the chiasma, or from great pressure in the third ventricle of the brain. Gummata and patches of sclerosis are by no means uncommon lesions in this position. Tumors of the pituitary body may in the first instance involve the decussating fibres of the commissure (as in acromegaly), or may be more extensive in their destructive effects.

THE THIRD NERVE.

The nucleus of the third nerve, or more correctly speaking the composite nucleus, part of which is common to both third nerves, is situated in front of the aqueduct of Sylvius and anterior to the anterior corpus quadrigeminum, while the posterior extremity almost reaches the nuclei of the fourth nerves. There are five distinct cell groups, and the

special group of cells subserving convergence, as well as certain of the other nuclei, send fibres to both nerves.

The two nerves emerge from the brain between the crura cerebri. Each nerve may be considered as consisting of two divisions, a superior division supplying the superior rectus and the levator palpebræ superioris muscles, and an inferior division supplying the internal rectus, the inferior rectus and the inferior oblique muscles. Through the ciliary ganglion the ciliary and the sphincter pupillæ muscles derive their supply from the third nerve.

The nucleus may be involved by tumors, sclerosis, hæmorrhages and encephalitis. The nerve may be affected by meningitis or gumma, and sometimes by aneurism. The close relationship of the two nerves at their origin render bi-lateral lesions common. Sometimes a neuritis, as in diphtheria, is responsible for paralysis of the muscles supplied by this nerve.

Symptoms of paralysis.—Where all the muscles supplied by this nerve are paralysed, the superior oblique and the external rectus are alone unaffected. The eyeball protrudes, there is divergent strabismus, ptosis, and the only movements possible are slight movements downwards, with rotation inwards due to the superior oblique, and outward movement due to the external rectus. The pupil is dilated owing to the dilator pupillæ muscle, which is supplied by the sympathetic acting unopposed, and accommodation is impossible owing to the ciliary muscle being paralysed.

In many cases the paralysis of the third nerve is only partial, and it is convenient to refer here to ptosis and paralysis of the iris, although many of the conditions in which these occur are only indirectly connected with the third nerve itself.

Ptosis.—This may be due to—(1) a lesion of the third nerve, as, for example, where the superior division is affected. (2) Congenital cases of ptosis are by no means

uncommon, and these cases are generally incurable. (3) There are cases of ptosis of cerebral origin, although it is difficult to say where the cortical centre is situated, and to explain in such instances how the ptosis occurs. (4) In idiopathic muscular atrophy, ptosis is not infrequently present along with involvement of other face muscles. (5) Closely associated with the last group may be mentioned cases in which in delicate females a ptosis may exist in the morning, and disappear in the course of the day. (6) Hysterical ptosis, generally bilateral, is not at all uncommon, and is usually accompanied by other typical phenomena of that disease. (7) A pseudoptosis is due to an involvement of the sympathetic fibres of the first dorsal root, and is the result of the sinking-in of the eyeball.

Paralysis of the Iris.—Paralysis of the iris should be divided into three groups:—(1) Loss of the light reflex or the Argyll Robertson phenomenon due to a lesion somewhere in the light reflex arc formed by the fibres of the optic nerve, the optic tract, the anterior corpus quadrigeminum, the fibres between that body and the sphincter pupillæ centre in the nucleus of the third nerve, and the motor limb passing by the third nerve to the sphincter muscle of the iris. Wernicke's pupil reaction has been described on page 1036. In testing for the presence of the Argyll Robertson phenomenon, a bright light should be used, and the patient should be told to look at a distant object, a useful procedure being to cover the eyes with the hands in such a way as to permit of the patient keeping them open (although covered with the hands), and then to uncover each eye separately. The use of a strong light renders the test more accurate. The Argyll Robertson phenomenon is present in most cases of locomotor ataxia, in which it forms one of the characteristic clinical features. It may also be found in most cases of general paralysis of the insane, and more rarely in certain cord

diseases. Its presence in cases of lesion of the optic tract has already been described on page 1036.

(2) Accommodation paralysis of the iris, or accommodation iridoplegia, is loss of contraction of the pupil on looking at a near object. The test can be easily applied by directing the patient to look first at a distant, and then at a near point.

(3) Loss of the skin reflex, or, in other words, loss of the typical dilatation of the pupil is obtained by pinching the skin of the forehead or neck. The value of this skin reflex is not very great, but it is desirable to investigate it in cases where there is spinal myosis.

Cycloplegia.—Cycloplegia, or loss of the power of accommodation, is due to paralysis of the ciliary muscle. When it is present the patient is unable to read or to see near objects clearly.

THE FOURTH NERVE.

The nucleus of the fourth nerve is situated immediately behind the third nucleus, and the nerve-fibres after leaving the nucleus decussate in the valve of Vieussens, pass round the outside of the crus, and go to the superior oblique muscle. A lesion which may involve the third nucleus may also damage the fourth, and the nerve is liable to be affected by meningitis, more especially if it be basilar.

Symptoms of paralysis.—The superior oblique muscle moves the eyeball outwards, downwards and slightly rotates it inwards. There is, as a result, defective movement in these directions, and double vision when the patient looks downwards and outwards, but, as a rule, there is little obvious strabismus unless when the movements usually carried out by the paralysed muscles are attempted.

THE SIXTH NERVE.

The nucleus is situated in the floor of the fourth ventricle, just above the striæ acusticæ, and the nucleus is partially surrounded by the fibres of the seventh nerve before they emerge from the pons. The sixth nerve makes its appearance just at the junction of medulla and pons and runs a long course, in which it is much exposed to pressure, until it reaches the orbit, terminating in the external rectus muscle. The nuclear origin of the opposite internal rectus muscle is connected with the sixth nucleus, and therefore in a nuclear lesion of the sixth there is paralysis of the corresponding internal rectus muscle.

A lesion of the sixth nucleus, owing to its close relationships to other nuclei and nerve-fibres, more especially those of the seventh nerve, generally implies a much more widespread paralysis than that due to the sixth nerve alone. The nerve is extremely liable to suffer from basilar meningitis, gummata and tumors.

Symptoms of paralysis.—Where the lesion is nuclear, as above stated, the opposite internal rectus is also paralysed. Where the nerve alone is injured the eyeball cannot be moved outwards, and there is double vision when the patient looks towards the paralysed side.

In examining paralysis of any ocular muscle or muscles, we should not merely know the extent and direction of movement, and the limitation of such movement owing to the loss of certain muscles, but, in addition, the degree of deviation of the pupil on the affected side. The *primary* and the *secondary* deviations must be examined so as to determine whether the strabismus is due to spasm or paralysis, and further it will help in distinguishing the muscles paralysed. *Primary* deviation is the deviation of the affected eye from the middle line when the patient fixes with both eyes a distant object. The *secondary* de-

viation is the deviation of the normal eye when the patient is forced to fix a distant object with the paralysed eye. These deviations must be measured and compared. The *secondary* deviation greatly exceeds the *primary* where the lesion is paralytic, while in cases of spasm, generally due to an error of refraction, the two deviations will be found to be equal. The existence of a false image is called diplopia. In every case of ocular paralysis it is desirable to ascertain the position of the false image, and this can be done by means of a piece of red glass. If the false image is on the same side as the eye with which it is seen, it is called simple or homonymous. If it is on the opposite side, it is crossed. The false image is projected in the direction in which the paralysed muscle ought to act, and its position will be further from the true image when the patient endeavours to use the paralysed muscle. The effect of erroneous projection is not infrequently giddiness, and sometimes nausea. The reader is advised to consult one or other of the special text-books on eye diseases for a further description of this important subject, a subject made considerably easier by means of diagrams.

The term *ophthalmoplegia* is frequently applied to paralysis of the ocular muscles, and *external ophthalmoplegia* is the term given to paralysis of those muscles which move the eyeball, while *internal ophthalmoplegia* implies paralysis of the internal muscles, in other words, the ciliary muscle and the muscles which contract and dilate the pupil. In many cases of ophthalmoplegia, syphilis will be found to be the cause of the condition.

Treatment of ocular paralysis.—Syphilis is often the cause of the paralysis, and antisyphilitic treatment should receive a careful and long-continued trial. In cases of locomotor ataxia it is difficult sometimes to arrest the degenerative change in the nerve, but in the case of a neuritis due to cold or rheumatism, counter-irritation

has sometimes been attempted. The counter-irritation can only, however, be applied at a considerable distance from the affected nerve. The forehead above the eyebrow or the skin behind the ear is usually selected. Electricity has been recommended in some cases, and strychnine should certainly be tried in diphtheritic, if not in other cases of ocular paralysis. Where all else fails it may be possible, by means of suitable glasses, to completely correct the double vision.

SPASMS OF OCULAR MUSCLES.

Conjugate deviation is a tonic spasm of ocular muscles, and is generally associated with cerebral hæmorrhage. The patient looks towards his lesion in most cases, whereas when the spasmodic element is specially marked, by which is implied the presence of considerable irritation, he looks away from the lesion. The cortical centre for conjugate deviation of head and eyes has been placed in the posterior portion of the middle frontal convolution, but whether this centre is often directly irritated or not, the connecting strands of nerve-fibres from it are probably accountable for the conjugate deviation referred to in these cases.

Spasms of ocular muscles may also occur in cases of meningitis, and in epilepsy and hysteria, tonic and clonic spasms are not uncommon. It remains, however, to mention the chief cause of ocular spasm, a cause which belongs rather to the domain of the specialist than to that of the physician. It is that in all cases of a serious error of refraction ocular spasm causes squint, and it does so most markedly in cases of hypermetropia.

Nystagmus is a clonic spasm of ocular muscles. It is usually lateral, sometimes vertical, and rarely rotatory. It may be constant or intermittent. It often occurs only when the weaker muscles are put on the stretch. Its presence may imply a lesion in the eyeball, and may have nothing

directly to do with any ocular muscle or its nerve supply. The rate of the spasms varies from a comparatively slow rhythmic series of movements to a very rapid, and almost uncountable rate, and the extent of the movements, often fairly large when the movements are slow, may be extremely fine where the movements are very rapid. The nucleus of Deiters is probably associated with nystagmus, and a lesion of it or of its connecting fibres may be responsible for the condition. Nystagmus occurs in a great variety of conditions, of which the chief are grouped together in the following table:—

1. Causes purely ocular, such as high grades of refractive error, choroiditis, keratitis, opacities in the lens, albinism, and other congenital malformations of retina and choroid.
2. Lesions in the pons, cerebellum, and optic thalami.
3. Insular sclerosis and hereditary ataxia.
4. Trade nystagmus, as in the case of miners, envelope folders, and other occupations in which the eyes are continuously being strained in some particular direction, or where rapid movements are made by the eyeballs for long periods of time.

Treatment.—The treatment of nystagmus depends entirely on the cause of the condition. Sometimes it can be removed by attention to the error of refraction, and in cases of trade nystagmus a long period of rest often effects complete recovery.

THE FIFTH OR TRIGEMINAL NERVE.

Before attempting the study of the affections of this nerve, it is desirable, in the first place, to revise and master the anatomy of the nuclei and the roots of the fifth nerve in the pons, and also to construct, however roughly,

a diagrammatic representation of the chief branches of the three divisions of the nerve, with their distribution.

Neuralgia of the fifth nerve, or *tic douloureux* has already been described on page 1005, and it is only necessary to refer here to paralytic lesions and masticatory spasms.

Etiology.—The lesion may be situated in the pons, in which position tumors, sclerosis or hæmorrhage is not uncommonly present, but it is rare to find the damage limited to the fifth nerve or its nuclei. In bulbar paralysis the nuclei of the fifth may be involved along with other nuclei in the medulla and pons. At the base of the brain, meningitis, syphilis, tumors, and caries of bone are common causes of lesion, but here, again, the fifth nerve does not suffer alone. The Gasserian ganglion may be involved by tumors and fractures, and in this case not merely will all three divisions of the nerve be involved, but probably the motor root will also be affected. The motor root does not form a part of the Gasserian ganglion, but lies behind it, and is therefore apt to be included in any lesion of the Gasserian ganglion. The first or ophthalmic division of the fifth nerve may be implicated by pituitary tumors, tumors pressing on the cavernous sinus, aneurisms of the internal carotid artery, or cellulitis in the orbit. The superior and inferior maxillary divisions of the fifth nerve may be invaded by tumors in the sphenomaxillary fossa.

Supra-nuclear lesions rarely involve the whole of the nerve, the motor fibres which join the third division of the nerve generally escaping, and therefore paralysis of the muscles supplied by the motor root of the fifth usually indicates a nerve lesion, and not a nuclear or a supra-nuclear one. A cortical lesion may cause paralysis of the muscles of mastication.

The fifth nerve supplies common sensation to the corresponding half of the face, the greater part of the side

of the head, the mucosa of the mouth, the tongue, and one nostril. The motor root of the fifth supplies the muscles of mastication, the masseter, temporal, and the two pterygoid muscles, and, in addition, the mylo-hyoid and posterior belly of the digastric muscles. Taste is supplied to the anterior two-thirds of the tongue by the chorda tympani nerve, which joins the lingual branch of the third division of the fifth, and, as indicated elsewhere, there is some doubt as to whether the taste fibres for the whole of the tongue (the chorda tympani fibres and those carried by the glosso-pharyngeal nerve) do or do not reach the brain by joining the second and third divisions of the fifth nerve.

Symptoms.—A lesion of the whole of the sensory portion of the fifth nerve causes hemianæsthesia of the face, and should there be any irritation, there may be pain, limited to the branch or branches of the nerve affected, and in addition herpetic eruptions may occur. The painful points referred to on page 1005 indicate the site where the branches of the nerve pass through bony foramina, and pressure over these may cause suffering. When the patient drinks he does not feel the half of the cup on the anæsthetic side, giving him the impression that the cup is broken, and eating is difficult owing to the food tending to accumulate between the anæsthetic cheek and the gum. He is apt to bite the cheek when eating, and sometimes the teeth loosen and decay. The nasal mucosa becomes dry, and smell may be affected, while the lachrymal and salivary secretions may be diminished. It is only where there is actual inflammation, and especially in the neighbourhood of the Gasserian ganglion, that trophic changes are apt to occur in connection with the eyeball, changes which may even end in corneal ulcer and panophthalmitis, but it should be remembered that in cases of supraorbital neuralgia, herpes frontalis, with keratitis and iritis, are by no means uncommon. It was

thought at one time that the integrity of the eye depended on preservation of the Gasserian ganglion, but the ganglion has been removed on many occasions without any trophic inflammation occurring in the corresponding eyeball. It is needless to add that in lesions of the fifth nerve a combination of anæsthesia with hyperæsthesia may occur, the latter indicating irritation which is likely to precede paralysis, and which will disappear should complete anæsthesia develop. Affections of taste are referred to separately.

Paralysis of the muscles of mastication may be recognised by the inability of the patient to chew food on the affected side, and by the flabbiness of the muscles when he attempts to clench the jaw. Three signs should be carefully noted which indicate a unilateral paralysis:—(1) the lower jaw cannot be moved towards the sound side; (2) when the lower jaw is depressed it is displaced towards the paralysed side; and (3) when the mouth is opened widely the condyle of the lower jaw markedly projects on the paralysed side.

Diagnosis.—The diagnosis is easy provided the reader has acquired an elementary knowledge of the distribution of the nerve. Where the Gasserian ganglion is responsible for the lesion, all three divisions, and generally the motor fibres, will be involved. Where the lesion is pontine it is apt to be bilateral and is rarely limited to the one cranial nerve. Where the lesion is in the internal capsule there is usually a complete hemianæsthesia.

Prognosis.—The prognosis depends largely on the nature of the lesion and the possibility of its removal.

Treatment.—The reader is referred for a description of methods of treating neuralgia to page 1008. Where syphilis is suspected potassium iodide should not be forgotten. Sometimes strychnine and sometimes electrical treatment

will be found helpful, especially when the muscles of mastication are paralysed.

SPASM OF THE MUSCLES OF MASTICATION OR TRISMUS.

Tonic spasms of the masticatory muscles occur in tetanus or lock-jaw. A more common but less severe form of the same kind of spasm is the leading feature in tetany. Tetany is not an uncommon accompaniment of dental neuralgia, although the spasm is generally slight and evanescent. The *clonic* type of spasm is one of the characteristic features of a rigor, and is well seen in the cold stage of a malarial attack. Tonic contraction of the masticatory muscles is a feature of the tonic stage of epilepsy, and it is followed by the clonic spasms of the same muscles, and to this the biting of the tongue is due. Both tonic and clonic spasms may also be met with in hysteria.

AFFECTIONS OF THE SENSE OF TASTE.

Although only the taste-fibres to the anterior two-thirds of the tongue are distributed by the lingual branch of the fifth nerve, it is convenient to refer here to alterations in taste generally. The reader is referred to page 1049 for a description of the course of the chorda tympani fibres in the Eustachian canal and their probable course from the geniculate ganglion to the brain. The course of the taste fibres for the posterior one-third of the tongue from the glosso-pharyngeal nerve upwards to the brain is open to considerable question. It is probable that the fibres pass by the *nervus intermedius* of Wrisberg, and perhaps the chorda tympani fibres may also reach the brain by the same nerve.

Taste and smell are closely dependent on each other. By taste we distinguish only saltness, sweetness, bitterness and acidity, and the relish which forms the chief pleasure

in eating a savoury dish depends on the integrity of the olfactory nerve-fibres. It is usual to find where smell is seriously interfered with, that the sufferer complains of loss of taste, inasmuch as he is deprived of gastronomic pleasures. Taste pure and simple can be tested by rubbing a small quantity of sugar, salt, quinine or vinegar on to the patient's tongue, care being taken to prevent the tongue being withdrawn into the mouth until the individual has written down on paper what he believes the substance used to be. A feeble galvanic current passed through the tongue gives a typical metallic taste, and this has been used as a method of testing the gustatory sense.

Ageusia is loss of taste, and the term should be limited to cases in which gustatory sense is abolished, and not merely to cases in which loss of smell has occurred.

Parageusia is perversion of the sense of taste. It may occur as an *aura* in epilepsy, but is more likely to be associated with insanity or hysteria.

THE SEVENTH NERVE.

The cortical centre is situated in the lower part of the ascending frontal convolution and the posterior part of the inferior frontal. This constitutes the cortical centre for the face. The fibres pass downwards through the internal capsule, and, with the exception of a few fibres which innervate the frontalis muscle, the orbicularis palpebrarum and some of the other muscles of the upper part of the face, they decussate in the middle of the pons and pass down to the seventh nucleus, which is situated in the lower part of the pons, just internal to the ascending root of the fifth nerve. The fact that these fibres which supply the frontalis muscle and other muscles of expression do not decussate explains the well-known fact that in an ordinary cerebral hæmorrhage in the neigh-

hood of the internal capsule these muscles are not markedly affected, while the muscles of the lower part of the face are paralysed. From the seventh nucleus, the seventh nerve forms a loop round the sixth nucleus, and appears just at the junction of the pons and medulla, close to the point of emergence of the eighth nerve. The seventh and eighth nerves pass close together to the internal auditory meatus, the seventh enters the Fallopian canal, and eventually emerges at the stylo mastoid foramen. In this part of its course the nerve has on it the geniculate ganglion, and between this ganglion and its point of emergence the nerve gives off a small branch to the stapedius muscle. This muscle antagonises the tensor tympani, and therefore, if it is paralysed, the tympanic membrane is kept on the stretch, and loud sounds are painful to the patient. The chorda tympani nerve joins the seventh nerve above the stylo-mastoid foramen,* and carries the taste fibres from the anterior two-thirds of the tongue to the geniculate ganglion, from which they either travel by way of the great superficial petrosal nerve to Meckel's ganglion and eventually reach the second division of the fifth nerve, or else they pass by some other route at present uncertain to the medulla.

Etiology.—Paralysis of the seventh nerve may obviously be due to a lesion in a number of different positions—(1) It may be cortical. (2) It may be in the internal capsule, or at all events above the middle of the pons. (3) It may be below the middle of the pons, and above the nucleus, in which case crossed paralysis will be produced—the arm and leg being paralysed on the opposite side of the lesion, and the face on the same side. (4) The lesion may be in the nucleus. (5) The lesion may be in the nerve itself, and

* The chorda tympani fibres enter by the *iter chordæ antierius* at the inner end of the Glaserian fissure, and run in the *iter chordæ posterius* to join the seventh nerve.

may involve the nerve either inside the cranial cavity, in the Fallopian canal, or just at or peripheral to the stylo-mastoid foramen.

Cortical lesions may be of the nature of tumors, softening, hæmorrhage, or possibly an abscess. In the internal capsule, hæmorrhage and less commonly thrombosis may occur. Thrombosis and embolism are of frequent occurrence in connection with the branches of the middle cerebral artery.

The nucleus may be affected by encephalitis, softening, hæmorrhage, or tumor, but as a rule other neighbouring nuclei are also affected.

The nerve may be involved at the base of the brain by meningitis, hæmorrhage or gumma, but will probably not suffer alone, the close relationship of the eighth nerve to the seventh implying its affection in almost every case. In the petrous part of the temporal bone, middle ear disease or fracture of the skull are the common causes of the lesion. In most cases, Bell's paralysis, by which is meant paralysis of the nerve, is due to rheumatism, sometimes to syphilis. A history of exposure to cold, especially a chilly blast on the side of the head, is not infrequently the antecedent history of a facial paralysis. Sometimes operative interference in connection with the glands of the neck, and especially the parotid, may result in a division of the nerve or some of its branches. Parenchymatous neuritis is the general form, although some cases are interstitial in nature.

A bi-lateral lesion is uncommon, but may be due to—(1) double ear disease; (2) a basal lesion, such as meningitis; (3) a lesion in the pons extending to both sides; or (4) a double cortical lesion.

Pathological Anatomy.—If the lesion is a *neuritis* it may be either parenchymatous or interstitial, and an account has already been given of each of these. If the lesion is *nuclear* in position, it may be an encephalitis, corresponding closely to poliomyelitis of the anterior horn cells, and

as a rule other nuclei are also involved. In some cases, as in the more chronic type of bulbar paralysis, the seventh nucleus suffers along with other neighbouring nuclei from the toxin, and it is rare to find the one nucleus alone affected. *Cortical* lesions include hæmorrhage, thrombosis, embolism and tumor. If the lesion involves the nucleus or the nerve, the muscles waste, and there are the marked electrical changes which constitute the reaction of degeneration, whereas, if the lesion is situated above the nucleus, the muscles only waste from disuse, and there are practically no electrical changes present.

Clinical features.—In Bell's paralysis, there is complete paralysis of the muscles supplied by the seventh nerve. The skin on the affected side is smooth. There is marked absence of wrinkles, and the eye cannot be closed because the levator palpebræ superioris acts unopposed. The patient, when he endeavours to close the eyes, rotates the eye on the affected side upwards so as to hide the pupil as far as possible under the upper lid. In time, there is marked drooping of the lower eyelid, which may result in a condition of ectropion, and the tears run down the cheek, due to the canaliculus of the lower lid not being properly opposed to the eyeball. Whistling, wrinkling of the face, moving the ear, are all much interfered with or rendered impossible on the affected side. The patient smiles with one side of his face, in drinking the lip cannot be properly opposed to the cup, and chewing becomes difficult owing to food accumulating between the cheek and the gum. He speaks indistinctly, and sniffing can only be performed with the one nostril.

If the lesion is situated outside the skull, or at all events before the chorda tympani fibres have joined the nerve, there is no interference with taste, whereas, should the chorda tympani fibres be affected, there is loss of taste in the anterior two-thirds of the tongue on the affected side.

Hearing is apt to become affected should the lesion be inside the petrous part of the temporal bone, as the auditory nerve generally participates in the affection; but it should be remembered that in the event of the branch to the stapedius being paralysed and the nerve of hearing intact, loud sounds become painful to the patient, as the tensor tympani acts unopposed. There should be no change in the soft palate or uvula, because the palatal muscles are not innervated by the seventh nerve.

It is of great importance to study the electrical reactions. In a well-marked case of Bell's paralysis there is loss of faradic excitability in the nerve and also in the muscle, while with the galvanic current what is called the reaction of degeneration develops (see page 839). The partial or incomplete reaction of degeneration may alone be present in a case of Bell's paralysis. The significance of these electrical reactions is obvious, when we remember that a partial reaction of degeneration is often associated with a paralysis only lasting from eight to twelve weeks, while a complete reaction of degeneration militates seriously against a favourable prognosis being given.

Pain is rare, because the nerve is a motor and not a sensory one, although it is true that occasionally a herpetic eruption appears along the line of the affected nerve. There may be some swelling of the affected part of the face.

In a certain number of cases contracture occurs, and generally where recovery has been long delayed and is incomplete. In such a case the affected side shows deep wrinkles, making it seem to the casual observer as if the paralysed side were the healthy one. This contracture is often accompanied by considerable pain.

A supranuclear lesion is fully described in connection with cerebral hæmorrhage: in certain cases where the lesion is cortical, Jacksonian epileptic seizures occur, and reference is made to this condition elsewhere.

Diagnosis.—There is no difficulty in diagnosing a case of Bell's paralysis, but it may be difficult to say exactly where the lesion is. Enough has been said to show how a knowledge of anatomy aids the physician in this matter. It is very rare to find the lesion involving the nucleus without other neighbouring nuclei also suffering.

Prognosis.—The greatest pains should be taken with the electrical reactions in a nerve palsy, because by no other means can an accurate guide to prognosis be obtained. A partial reaction of degeneration indicates a rapid recovery.

Treatment.—Counter-irritation is certainly one of the best means of treatment for interstitial neuritis, either fly blisters or the application of the button cautery as near the stylo-mastoid foramen as possible. It is doubtful whether counter-irritation is so useful in parenchymatous neuritis. In syphilitic cases iodide of potash should be administered, and possibly, in some cases, mercury. As soon as the inflammatory changes in the nerve have subsided, electrical treatment should be commenced, the positive pole being applied behind the ear, and the negative to the affected muscles, and massage should not be neglected. The ear should be examined carefully and suitable treatment undertaken where there is middle ear disease.

Surgery has assisted greatly in the recovery of certain cases incurable by other means, the seventh nerve below the level of the lesion being sutured either to the spinal part of the spinal-accessory nerve or to the hypoglossal nerve, and in time it is possible for the functions of the seventh nerve to be taken up by a new group of nuclear cells and a new part of the cerebral cortex. The only objection to the use of the spinal-accessory for this purpose is that when the patient shrugs his shoulder, the energy may overflow into the seventh nerve and cause a grimace to occur.

SPASMS OF THE SEVENTH NERVE.

Spasms of part or of all the muscles innervated by the seventh nerve are common in many conditions. They may be unilateral or bilateral, and many of the habit spasms so common in children and nervous people belong to this group.

The spasm may have a definite organic origin. It may be due to a carious tooth or to some more distant source of irritation, such as the presence of worms. It is often highly infectious, one child starting the habit in a number of other children who are more or less susceptible.

It is most frequently the muscles of the eye which are affected, in other words, it is often blepharospasm, sometimes it is winking along with twitching of neighbouring muscles of the face. These spasms are very troublesome, are very difficult to cure, and may become a lifelong habit of the patient. The treatment should be directed to the removal of any irritation, dental or otherwise, and the presence of any painful point in the realm of the fifth nerve should be noted, and the part treated by counter-irritation or other measure. In some cases freezing the cheek has been found helpful, and it is only in rare instances that section of the nerve is called for.

THE EIGHTH OR AUDITORY NERVE.

The eighth nerve consists of two parts, the cochlear and the vestibular, of which the former is the nerve of hearing, the latter the nerve which has to do with equilibration.

1. *The Cochlear Nerve.*—The auditory centre is situated in the superior temporo-sphenoidal lobe, and occupies the posterior two-thirds of that lobe. The auditory centre in the left side of the brain in a right-handed person is the word-hearing centre, and is one of the most important of all our cerebral centres, a lesion of this centre not merely rendering the patient unable to understand symbols heard

with the ear, but generally rendering him absolutely aphasic and unable to utilise his other mental centres which may be intact. The cochlear fibres pass downwards from the temporo-sphenoidal lobe, a little behind the posterior part of the internal capsule, on their way to the cochlear nucleus. These fibres have connections with the posterior corpora quadrigemina and the internal geniculate bodies, and in this way the fibres which conduct auditory impressions come into close relationship with those which subserve sight. Tumors, hæmorrhage, thrombosis and, more rarely, abscess may cause lesions of the auditory cortical centre.

At the base of the brain the eighth nerve is closely related to the seventh, and is liable along with the other cranial nerves to be involved by tumors, meningitis, hæmorrhage and other lesions, and in this position the vestibular portion of the nerve suffers in addition.

Deafness is often due to a fault in the passages, either the outer ear, tympanum, or middle ear, and not to any nerve defect. It must be remembered that auditory hyperæsthesia is not infrequently associated with a marked degree of deafness, and tinnitus aurium is the usual term applied to denote the results of auditory hyperæsthesia.

Tinnitus Aurium.—This consists in sounds of very varying character heard by the patient, and which resemble hissing, whistling, chirruping, humming, throbbing or other sounds. They are not infrequently the result of some change in the tympanic membrane, either thickening of the drum or the presence of wax pressing on it. In other cases they are the result of the patient hearing the blood rushing through his arteries and veins, and certainly the arteries are not infrequently responsible in cases of atheroma, while the veins are more likely to be the causal factor in anæmia. In other cases, some not very explicable changes in the labyrinthine fluid may permit of the labyrinthine circulation being heard by the patient, and it is probable that

quinine and similar drugs cause cinchonism in this way. Choreic twitching of the stapedius muscle has also been considered to be a cause of tinnitus, and an effusion into the middle ear is sometimes responsible for gurgling sounds heard by the patient. Not infrequently the semicircular canals are involved along with the nerve of hearing, and in describing Ménière's disease reference will be made to the constant association of tinnitus aurium with it.

There are cases where the patient has hallucinations of hearing, but these are not true instances of tinnitus aurium and are really insane delusions.

2. *The Vestibular Nerve.*—A lesion of the vestibular nerve, the vestibular nucleus, the semicircular canals, or the connections with the cerebellum and other parts of the brain cause vertigo, loss of equilibration, nystagmus and interferences with the co-ordination of certain groups of muscles. The exact clinical symptoms present are dependent on the nature and extent of the lesion.

MÉNIÈRE'S DISEASE.

This is a lesion of the semicircular canals, and may be due to a variety of different pathological conditions. Amongst the commoner but at the same time the most serious of these are hæmorrhages or inflammatory exudations, and among the more trivial causes are the pressure of wax on the tympanic membrane, and anything which may interfere with the intra-labyrinthine pressure. One of the forms of aura in epilepsy may be an auditory vertigo.

Etiology.—A common cause of hæmorrhage is a loud noise, such as the firing of a heavy piece of ordnance, or prolonged exposure to exceedingly loud sounds, as in the case of riveters working inside metal boilers. It is probable that the actual hæmorrhage depends on some other causal factor being in operation. In leucocythæmia such hæmor-

rhages have also been noticed. Interference with the drum may be due to excess of wax in the outer ear, which may be made to press on the tympanic membrane during the process of syringing for its removal. Forceful syringing, where the jet of water impinges with violence on the delicate tympanic membrane, may of itself be sufficient to produce auditory vertigo. Apart from these causes of auditory vertigo, there are undoubtedly isolated instances of Ménière's disease for which no satisfactory explanation can be given.

Symptoms.—The typical symptoms are (1) vertigo, (2) vomiting, and (3) tinnitus aurium.

Vertigo may come on suddenly, causing the patient to fall, or it may be continuous, and prevent the sufferer from even sitting up in bed. It is frequently associated with sickness and vomiting, which continue so long as the patient endeavours to sit up or stand. The tinnitus aurium is often very severe. Along with these typical phenomena there may be nystagmus, the patient may become deadly white, beads of cold clammy perspiration breaking out on his forehead, and, where the attacks are paroxysmal there may be a brief period of unconsciousness.

Diagnosis.—There are other forms of vertigo more or less easily differentiated. Ocular vertigo is due to double vision from ocular paralysis, but in this case the giddiness disappears when the patient closes one eye or shuts both. The vertigo of gastric origin is not, as a rule, associated with deafness or other auditory phenomena, is generally of a passing nature, and is directly due to some indiscretion of diet. Cardiac vertigo is generally very different in history, and the feeble heart sounds with evidence of serious cardiac lesion are usually amply sufficient to prevent any mistake being made. Hysterical cases simulating Ménière's disease are not infrequent, and can generally be

distinguished by the presence of typical hysterogenetic areas and other definite functional features of hysteria or neurasthenia.

Prognosis and Treatment.—Trivial cases, such as those due to wax, will yield to treatment, but where a hæmorrhage has occurred, the patient may remain a constant sufferer from this form of vertigo, and the advisability of attempting by operative interference to remove the semicircular canals should at least be carefully considered. There is no reason why this should not be done where the patient is physically capable of standing an operation. The remedies usually administered are sedatives, such as one or other of the bromides. Needless to say, in syphilitic cases potassium iodide should be given. Where there is arterial degeneration, nitroglycerin is often found helpful. In every case it is desirable to eliminate the possibility of the vertigo being of ocular origin, and any error of refraction or astigmatism must be carefully excluded.

THE NINTH OR GLOSSO-PHARYNGEAL NERVE.

It is impossible to dissociate this nerve from the tenth so far as the nuclear origin of both is concerned. The nerve carries sensory fibres to the upper region of the pharynx, and it should be remembered that by it the taste fibres to the posterior third of the tongue and the neighbouring portion of the fauces are also carried. The motor fibres of the nerve go to the stylo-pharyngeus muscle and the middle constrictor of the œsophagus.

The nerve may be paralysed either from a lesion in its course or from an involvement of its nucleus, such as occurs in bulbar paralysis. The commonest lesions of the nerve are the result of meningitis or tumor,

Symptoms.—The upper portion of the pharynx is anæsthetic, and owing to the involvement of the middle constrictor muscle there is difficulty in the act of swallowing food, while loss of taste in the posterior third of the tongue may also be present.

THE TENTH. VAGUS OR PNEUMOGASTRIC NERVE.

The remarks already made about the close association of the ninth and tenth nuclei should be remembered, and it is usual in diagrammatic sketches of these nuclei to represent a separate sensory and a separate motor nucleus situated in the medulla, both of which are common to each nerve.

Etiology.—The nuclei of the vagus are often involved in cases of bulbar paralysis, in which case the glosso-pharyngeal nerve will also suffer. The nerve may be affected by meningitis, tumors and aneurisms, as well as by wounds in the region of the neck. The nuclear cells as well as the nerve-fibres—in other words the whole neurones—may suffer in cases of toxic neuritis, the result of alcohol or diphtheria. Neuromata have sometimes been described as occurring on this nerve, and may naturally interfere with its functions.

Symptoms.—It is a hopeless task to attempt to describe in detail in a work such as this all the branches and all the functions carried out by these branches of the vagus nerve. Reference will therefore be only made to the more important of these branches.

(1) *Branches to the Pharynx.*—The vagus along with the glosso-pharyngeal nerve supplies sensation to the pharyngeal mucosa and motor power to the pharyngeal muscles. A

lesion of the nerve causes difficulty in swallowing, and the food may stick in the throat, tending to induce spasm, and if the soft palate is also paralysed (probably supplied by the spinal accessory through the vagus) fluids may pass down the nose. Spasm of the pharyngeal muscles is not uncommon in cases of hydrophobia and some other diseases.

(2) *Branches to the Larynx*.—The intrinsic muscles of the larynx are supplied by nerve-fibres which originate in the medullary nucleus of the spinal accessory, but are mainly distributed through the recurrent laryngeal branch of the vagus. It is therefore appropriate to describe laryngeal paralysis in connection with this nerve. The superior laryngeal branch of the vagus is the sensory nerve for the mucous membrane of the larynx, and supplies also one muscle, the crico-thyroid. The recurrent laryngeal nerve supplies all the intrinsic laryngeal muscles. The course of the right and left recurrent laryngeal nerves must be remembered, and especially the relationship of the left nerve to the arch of the aorta. The left recurrent laryngeal nerve may therefore be involved by aneurism of the arch of the aorta, whereas the right recurrent laryngeal is more apt to be affected by pleuritic adhesions in connection with the right lung. There are two types of laryngeal paralysis other than that due to an affection of the whole recurrent laryngeal nerve itself, viz., adductor and abductor paralysis. In cases of catarrh of the larynx, and also in cases of hysteria, the paralysees do not, as a rule, endanger life. In other words, the adductors of the glottis, or the tensors of the cords, or the arytenoideus muscle may be affected, whereas in diphtheria the abductors of the glottis are more likely to suffer. In recurrent laryngeal paralysis or paralysis of the one cord the abductor generally suffers first, thus causing considerable spasm, and only later do the adductors suffer, bringing the cord eventually into the so-called "cadaveric" position. The following types of paralysis may be described :—

(a) *Adductor Paralysis*.—This is due to the paralysis of the crico-arytenoideus lateralis muscle, or the arytenoideus muscle, or both together. It is a common result of catarrh of the larynx, and is not uncommon in hysteria. The cords cannot be brought into apposition, the one on the affected side being markedly abducted by the unopposed abductor muscle. The patient can breathe freely, but is aphonic. Paralysis of the arytenoideus muscle alone, implies the leaving of a triangular-shaped opening situated posteriorly during attempted phonation.

(b) *Abductor Paralysis*.—This is paralysis of the crico-arytenoideus posticus muscle, and it is a common form of diphtheritic paralysis. It may occur in bulbar paralysis, and it should be remembered that the paralysis of this muscle is the initial result of complete recurrent laryngeal paralysis. The patient has difficulty in breathing, and if both muscles are involved, asphyxia is imminent. There is almost invariably stridor in breathing.

(c) *Paralysis of the whole Recurrent Laryngeal Nerve*.—At first the affected cord remains almost in the middle line, and only later is it found in its typical "cadaveric" position, midway between full abduction of the cord and the middle line. Breathing, which at first may be somewhat noisy because of the abductor paralysis, soon becomes freer, but speech is difficult owing to the inability to approximate the two cords, and as a rule the unaffected cord endeavours to cross the middle line to approximate with the paralysed cord, and so help in phonation.

Spasm of the muscles of the larynx has already been described under laryngismus stridulus. The laryngeal crises in locomotor ataxia are due to laryngeal spasm.

(3) *Branches to the Heart*.—The cardiac plexus derives its branches both from the vagus and also from the sympathetic. The vagus retards, the sympathetic accelerates the cardiac contractions. Irritation of the vagus will increase the inhibition, whereas the paralysis of one vagus

ought to be followed by an acceleration of the heart's action. It is probable, however, that both vagi require to be affected before much acceleration results.

(4) *Branches to the Lungs.*—The pulmonary plexus consists of branches from the vagus, and also from the sympathetic. Pressure on the root of the lung causes septic pneumonia, which is sometimes called vagus pneumonia, although we cannot definitely say whether the vagus branches are entirely responsible for the incidence of the pneumonia. It should also be stated, that as the vagus supplies the muscles of deglutition as well as the sensory and motor nerves of the larynx, food may be inspired into the bronchial tubes, and in this way a true vagus pneumonia often results. Probably the terminations of the vagi nerves in the bronchial tubes are the cause of the act of coughing when the irritant is situated in the bronchi.

(5) *Branches to the Stomach and Œsophagus.*—Paralysis of the œsophageal branches causes difficulty in swallowing, while œsophageal spasm—the result of tumor, stricture or some functional condition—is unquestionably due to the vagus nerve.

The vagus supplies sensory and motor fibres to the stomach, and gastric pain and cramp are unquestionably of pneumogastric origin, while the peristalsis, to which the mixing of the food in the stomach is largely due, is also dependent on the tenth nerve.

We have here merely enumerated a few of the chief and more important branches and functions of the vagus nerve, but the account is full enough for most practical purposes. Sometimes one or other group of branches may be more affected, in other cases all the functions of the vagus may suffer, when death is an inevitable result.

Treatment.—Laryngeal paralysis can be treated by electricity or by massage. One pole should be applied to each side of the larynx, and either galvanism or faradism

may be used. Where abductor paralysis is bilateral, tracheotomy should be performed so as to avoid risk of asphyxia.

THE ELEVENTH OR SPINAL ACCESSORY NERVE.

The spinal portion of the nerve supplies the trapezius muscle in part and the sterno-mastoid, while the accessory or medullary division innervates the intrinsic muscles of the larynx. The medullary nucleus of the nerve is situated just below the ninth and tenth nuclei, while the nerve-cells representing the spinal centre are situated in the cervical portion of the cord. The nucleus in the medulla may be involved in cases of bulbar paralysis, and the spinal portion in various cord diseases, particularly in progressive muscular atrophy. The nerve may suffer from injuries in the region of the neck, especially to the cervical vertebræ, while tumors occasionally implicate it.

Paralyses of the laryngeal muscles have already been described. Where the sterno-mastoid and the trapezius—so far as it is supplied by this nerve—are paralysed, the patient cannot shrug the shoulder on the affected side, nor can he turn his head towards the opposite side of the body. The shoulder droops, and owing to the rhomboids and the levator anguli scapulæ being unantagonised the scapular angle is rotated inwards. When the arm is raised the scapula moves as a whole, the trapezius failing to act as a fixing agent so as to permit of the leverage action of the deltoid.

If both sterno-mastoids are paralysed, the head may fall back, although if the trapezii are also both involved the head tends to fall forward, and the attitude of the patient gives the typical picture of an advanced case of the disease.

in which this paralysis generally occurs, namely, progressive muscular atrophy. Twisting of the spine is common in cases where paralysis of the eleventh nerve is unilateral.

The paralysed muscles become soft and flabby and the reaction of degeneration may be obtained. As it is probable that the soft palate is innervated by fibres from the medullary nucleus, when this nucleus is affected the speech becomes nasal and fluids drunk are apt to regurgitate down the nose.

Diagnosis.—The diagnosis is easy, and the prognosis depends much on whether the lesion involves the nerve or the nuclear centres; if the latter, it is not so favourable.

Treatment.—Treatment must be carried out on general lines. Counter-irritation, massage and electrical treatment may be helpful when the lesion is in the nerve. Where, on the other hand, the nuclei are involved, the treatment indicated under bulbar paralysis and progressive muscular atrophy should be prescribed.

SPASM OF THE MUSCLES SUPPLIED BY THE ACCESSORY PORTION OF THE SPINAL ACCESSORY NERVE—TORTICOLLIS OR WRY-NECK.

In most cases of torticollis other muscles than those referred to above become affected, but the sterno-mastoid and the trapezius are the muscles mainly affected.

The Congenital form of torticollis is due to shortening of the sterno-mastoid on one side. Some authorities believe that it is due to traction on the head of the child at birth, but whether this explanation be sufficient or not, the affected muscle is shortened and markedly atrophied. Another suggestion has also been made, that the lesion is similar to

what occurs in infantile paralysis when it causes talipes. Tenotomy affords relief to the condition.

The Spasmodic form of torticollis is of two types—*tonic* and *clonic*. The sterno-mastoids and other neck muscles are affected. Amongst the causes commonly assigned for these conditions are blows on, and injuries to the side of the neck, exposure to cold, and that important if somewhat vague element in the family history of certain patients which may be expressed by the term *neurotic*.

In the *tonic* type the sterno-mastoid is contracted, drawing the occiput back towards the affected side, and the chin is tilted upwards. Other muscles may participate, and especially the trapezius. Pain is often associated, and the spasm may persist so long that hypertrophy of the muscles results.

The *clonic* type is much more widespread, many other muscles being associated on the affected side along with the sterno-mastoid. Not merely does the trapezius contract, but also the splenius and the platysma myoides. These clonic movements are very continuous and harass the patient, although they cease during sleep. They are increased by nervousness, and give rise to a considerable amount of pain.

Prognosis.—The prognosis is fairly good in *tonic* cases, but is very unfavourable in those which are *clonic*.

Treatment.—The treatment consists in the administration of bromides and other sedatives so as to soothe the spasm, and in *tonic* cases the spinal portion of the nerve may be divided, although, unfortunately, in the *clonic* type, nerve section has not resulted in the benefit which might have been anticipated.

THE TWELFTH OR HYPOGLOSSAL NERVE.

This nerve supplies the muscles of the tongue, not merely the intrinsic but also the extrinsic muscles by which the tongue is protruded. The nucleus of the nerve may be affected in cases of bulbar paralysis, and a supranuclear lesion is commonly situated in the region of the internal capsule, and forms part of an ordinary hemiplegia. The nerve may be affected by meningitis, tumor or other lesion after its emergence from the medulla, and in its passage towards the tongue muscles, injuries and wounds are sometimes responsible for the paralysis.

Symptoms.—Where the lesion is supranuclear, there is no wasting of muscles on the affected side, whereas if the lesion is nuclear or infranuclear, there is wasting and sometimes fibrillary twitchings. In supranuclear lesions, although the tongue may be protruded towards the paralysed side for some time after the hæmorrhage, recovery to a great extent is usual, and speech is rarely seriously affected. On the other hand, in nuclear or nerve lesions, speech is difficult and recovery of movement depends on the removal of the causal factor. In these lesions, too, the wasted tongue, shrivelled and shrunken on the paralysed side, is very typical, and in bilateral paralysis, which is usually of nuclear origin, the tongue cannot be protruded from the mouth, speech becomes almost impossible, and deglutition is rendered difficult by the loss of the assistance of the tongue in moving the bolus of food towards the pharynx.

Diagnosis.—The diagnosis consists largely in an attempt to localise the position of the lesion. Sufficient data have been given to enable the reader to differentiate between supranuclear, nuclear and nerve lesions.

Prognosis.—The prognosis is favourable in supranuclear lesions, unfavourable in nuclear, and only favourable in nerve lesions if the cause of the condition can be successfully treated by surgical or other measures.

Treatment.—Potassium iodide may be given where a specific cause is present, while electrical treatment applied to the affected muscles is sometimes of use.

SPASM.

Spasm of the muscles of the tongue is one of the causes of stuttering, and it may also occur in chorea, epilepsy and other nerve affections.

Section 9.

DISEASES OF THE MUSCLES.

I. MYOSITIS.

DEFINITION.—Inflammation of the voluntary muscles.

Etiology.—The inflammation may be caused by germs, animal parasites and toxins which generally attack the muscles secondarily. But the myositis may be primary. A suppurative form, due to the staphylococcus aureus and allied pyogenic germs, is described as not infrequent in Japan. It usually sets in suddenly with fever and prostration and ends in abscess formation among the muscles, which may result in general pyæmia. A non-suppurative form is described under the name of dermato-myositis as occurring somewhat widely in America and elsewhere. The muscles of the arms and legs become swollen in places, tender, firm and hard, and the skin is inflamed over them. It may cause much interstitial change. It has been spoken of as a pseudo-trichinosis from its resemblance to true trichinosis, from which it can be distinguished only by removing a portion of an affected muscle. Gummata and trichinosis cause a localised myositis. A hæmorrhagic form of interstitial myositis is also described.

II. MYOSITIS OSSIFICANS PROGRESSIVA.

A rare chronic disease in which the majority of the skeletal muscles of the body become transformed into bone.

It begins as a swelling of the muscles of the neck or back, which may be accompanied by fever. The skin may be red. The swelling becomes gradually indurated and gives place to bone.

III. MYOTONIA CONGENITA—THOMSEN'S DISEASE.

DEFINITION—A peculiar transient cramp of parts of voluntary muscles on attempting voluntary movements. It was first described by Thomsen, who himself suffered from it, and in whose family it had existed for five generations.

Etiology.—It occurs in several members of a family for several generations. Males suffer more than females. It is rare on the whole, but is commoner in Germany, Norway and Sweden than elsewhere.

Morbid Anatomy.—Hypertrophy of muscular fibrils, proliferation of nuclei and indistinct transverse striation are described as being present in the muscles.

Symptoms.—The disease begins in childhood and shows itself as a stiffness and slowness in every voluntary muscular movement at the commencement. Both contraction and relaxation are slow on account of a tonic spasm which comes on when the patient wills to do any muscular effort. Thus in walking, one leg is put forward slowly, and halts a little before finishing the step. The next few steps are the same and then become normal and may remain so for hours without any return of the spasm. After a rest, however, the same slowness in beginning to walk again occurs. Other movements are affected in the same way. The patient rises from a chair slowly. The muscles of the leg are most affected, those of the arms more than those of the face, which are very slightly touched. Cold, damp, heat and excitement generally increase the spasms.

Tapping a muscle causes a groove and the reaction to faradic electricity is increased. Erb's myotonic reaction is generally present, *i.e.*, the muscles under both galvanic and faradic stimulation attain their maximum contraction slowly and relax slowly, wave-like contractions passing from the cathode towards the anode. The disease is incurable but may be temporarily arrested. It rarely does more than cause the patient slight inconvenience, and does not tend to get worse as age advances.

The Treatment requires no reference because nothing can be done, although as a rule the patient learns how to prevent the spasms coming on by careful management, mainly directed to the slow initiation of any muscular movements.

INDEX.



- Abasia, 989.
 Abdomen, tapping, 722.
 Abdominal muscles in acute peritonitis, 761, 762
 „ reflexes, 840.
 Abducens nerve, 1040.
 "Aberrant rests" of Grawitz, 827.
 Abortion in acute yellow atrophy, 727.
 „ relapsing fever, 54.
 „ small-pox, 152, 155.
 „ syphilis, 189.
 „ absence of, in typhus fever 197.
 Adenia, 374.
 Abscess, actinomycotic, 130
 „ alveolar, 613.
 „ appendicular, 689
 „ atheromatous, 479
 „ biliary, 743.
 „ brain, 893.
 „ brain in bronchiectasis, 513
 „ brain in middle ear disease, 893.
 „ cerebellar, 894, 956.
 „ cold, 119.
 „ dysentery, 209.
 „ filaria bancrofti, 270.
 „ gastric ulcer, 656.
 „ glanders, 221.
 „ hepatic, 219, 536, 710, 711, 743.
 „ influenza, 560.
 „ ischio-rectal, 680
 „ kidney, 821.
 „ lung, 549, 560, 591.
 „ malignant endocarditis, 440
 „ myocarditis, 422.
 „ pancreas, 751.
 „ perinephric, 828.
 „ pneumonia, 536.
 „ psoas, 119, 900.
 „ pyæmic, 137.
 „ retropharyngeal, 617, 920.
 „ stomach, 649.
 „ subphrenic, 657, 765.
 „ tropical, 711.
 „ tubercular, 119.
 Abyssinians and tapeworms, 258
 Acarus scabei, 272
 Accelerator nerve of heart, 1061.
 Accentuated aortic second sound in aortic stenosis, 455.
 „ nephritis chronic interstitial, 815.
 Accentuated pulmonary second sound in mitral incompetence, 447
 „ mitral stenosis, 450
 Accessory adrenals, 401, 403.
 „ thyroids, 391 397
 Acetone in diabetes mellitus, 333, 334
 „ urine, 792
 Achondroplasia, 1003
 Acid fast bacilli, 112
 Acne from bromides, 976
 Acromegaly, 1001.
 Actinomyces, 129
 Actinomycosis, 129
 „ abscesses, 130
 „ appendical, 131.
 „ blood spread, 131
 „ bronchitis, 130.
 „ cæcum, 131
 „ cattle 130
 „ cells, 130.
 „ cereals, 129
 „ colonies in pus, 131.
 „ fever, 131.
 „ intestine, 131, 697.
 „ liver, 131, 697, 711.
 „ lung, 131, 591.
 „ micro-organism, 129
 „ mouth, 130, 614
 „ ovary, 697.
 „ potassium iodide, 132.
 „ skin, 131.
 „ sinuses, 130.
 „ suppuration, 130, 591.
 „ vermiform appendix, 131.
 „ visceral, 132.
 „ woody tongue, 130.
 Acupuncture in sciatica, 1007.
 Acute bulbar paralysis, 918,

- Acute yellow atrophy, 724.
 „ acute phosphorus poisoning, 724.
 Addison's disease, 402.
 „ asthenia, 404.
 „ hæmatoporphyrinuria, 786.
 „ loss of internal secretion, 403, 404.
 „ pigmentation, 35, 404, 405.
 „ symptom, complex, 403, 404, 405.
 „ thymus gland, 397.
 „ tuberculosis, 402, 403.
 „ vomiting, 404.
 Adenoids, 495, 622.
 „ cleft palate, 495.
 „ cough (barking or stomach), 495.
 „ deafness, 495, 623.
 „ mouth breathing, 495.
 „ tonsillar hypertrophy, 622.
 Adherent pericardium, 416.
 Adrenalin, 401.
 Adrenal gland, 401.
 „ accessory, 401, 403.
 „ destruction, 403.
 Adrenal rests, 401.
 Ægophonic vocal resonance, 603.
 Aerogenic tubercle, 564.
 Æstivo-autumnal or summer-autumn fevers, 238.
 „ parasite, 244.
 Affrent or sensory system, 832.
 African horse sickness, 234.
 Ageusia, 1048.
 Agglutination test in glanders, 221.
 „ typhoid fever, 68.
 Agoraphobia, 992.
 Agraphia, 959.
 Ague, 238.
 Air hunger in diabetes mellitus, 334.
 Air, open, treatment of phthisis, 122, 583.
 Albuminuria, 787.
 „ alcoholism, 275.
 „ cardiac disease, 447, 788.
 „ cholera, 83.
 „ congestive, 788.
 „ cyclical, 788.
 „ diabetes, 332, 335.
 „ diphtheria, 788.
 „ erysipelas, 141.
 „ lead poisoning, 219, 788.
 „ liver, acute! yellow atrophy, 726.
 „ measles, 788.
 Albuminuria—
 „ meningitis, epidemic cerebro-spinal, 78.
 „ neurotic, 788.
 „ pathological, 788.
 „ pneumonia, 537, 538, 788.
 „ physiological, 787.
 „ renal disease, 778, 788, 803, 808, 815.
 „ scarlet fever, 165-168, 788.
 „ significance, 789, 804.
 „ small-pox, 151.
 „ syphilis, 788.
 „ toxic, 788.
 „ tuberculosis, pulmonary, 569.
 „ typhoid fever, 788.
 „ typhus fever, 197.
 „ yellow fever, 203, 204.
 Albumoses, 629.
 Albumosuria, 789.
 „ empyema, 789.
 „ myelopathic, 789.
 „ pneumonia, 789.
 Albuminuric retinitis, 804, 1031.
 Alcaptonuria, 793.
 Alcohol in treatment of fevers, 199, 200.
 Alcoholism, 274.
 „ acute, 274.
 „ albuminuria, 275.
 „ amaurosis, 1035.
 „ amblyopia, 1035.
 „ apomorphin, 277.
 „ brain, wet, 853.
 „ cardiac failure, 278.
 „ chronic, 275, 278.
 „ collapse, 274.
 „ coma, 274.
 „ convulsions, 274, 277.
 „ cure (gold), 278.
 „ delirium tremens, 276, 277, 278.
 „ drowsiness, 275.
 „ facies, 275.
 „ fatty degenerations, 26, 32, 277.
 „ fever, 276, 277.
 „ fibrosis, 277.
 „ gastric catarrh, 638, 642.
 „ hallucinations, 276.
 „ hypnotism, 278.
 „ illusions, 276.
 „ insomnia, 275, 276, 278.
 „ irritability, 275.
 „ liver enlargement and cirrhosis, 275, 714.

Alcoholism—

- " meningitis, 853.
- " narcosis, 275.
- " nephritis, 798.
- " neuritis (multiple), 275.
- " obesity, 277.
- " pneumonia, 539, 541.
- " spleen, 275.
- " syncope, 274, 277.
- " tremors, 275, 276.

Algid form of yellow fever, 203.

- " stage of cholera, 83.

Alimentary glycosuria, 334.

Alkaloids, animal, in food poisoning, 279.

Allocheiria, 933.

Alopecia in syphilis, 185.

Alternate paralysis, 955.

Altitudes, high, and hæmorrhage, 494, 529.

Altitudes, high, and treatment of tuberculosis, 583.

Aluminosis, 556.

Amaurosis, 1035.

- " alcohol, 1035.
- " hysteria, 988.
- " lead, 1035.
- " quinine, 1035.
- " toxic, 1035.
- " uræmic, 783, 816.

Amaurotic family idiocy, 927.

Amblyopia, 1035.

- " tabes dorsalis, 935.

Ambulatory typhoid fever, 67.

Amœba in liver abscess, 711.

Amœbic dysentery, 207.

Amnesia verbalis, 961.

Ampho-peptones, 629.

Amphoric breathing, 580.

Anyloid degeneration, 29, 30.

- " intestine, 29, 576, 678
- " kidney, 29, 773.
- " liver, 29, 707.
- " lymphatic glands, 30.
- " phthisis, 30, 576.
- " spleen, 29.
- " supra-renals, 30.
- " syphilis, 30, 188.

Anylopsin, 629.

Amyotrophic lateral sclerosis, 913, 914.

- " syringomyelia, 863.

Anæmia, 347.

- " ankylostomiasis, 265, 353.
- " bothriocephalus latus, 258, 353.
- " brain, 865.

Anæmia—

- " brain in aortic incompetence, 452.
- " cancer, 347.
- " chlorosis, 349.
- " dropsy, 7.
- " gastric cancer, 665.
- " gastric catarrh, chronic, 645.
- " gastric ulcer, 662.
- " infantile, 370.
- " lead poisoning, 290.
- " leukæmia, 361, 368.
- " lymphadenoma, 377, 378.
- " malaria, 242.
- " Malta fever, 88.
- " mitral incompetence, 446.
- " peritonitis, chronic, 769.
- " pernicious, 352.
- " post-hæmorrhagic, 348, 358.
- " primary, 347, 349.
- " renal disease, 776, 804, 808, 816.
- " rheumatic fever, 300.
- " sclerosis of cord, 943.
- " scurvy, 385.
- " secondary, 347, 358.
- " splenic, 370.
- " spinal cord, 866.
- " syphilis, 187.
- " trichocephalus dispar, 254.
- " tuberculosis, 119, 579.
- " typhoid fever, 61, 64.

Anæmic murmurs, 351.

Anaerobic bacillus of tetanus, 101.

Anæsthesia—

- " Brown-Sequard's paralysis, 844.
- " dolorosa, 900.
- " hysteria, 988.
- " leprosy, 127, 864.
- " Morvan's disease, 864.
- " tabes dorsalis, 933.

Anarthria, 960.

Anasarca, 5.

Aneurism, 481.

- " abdominal aorta, 481.
- " atheroma, 479.
- " cerebral arteries, 481, 869, 888.
- " carotid, 481.
- " carotid (internal) pressing on first branch of fifth nerve, 1044.
- " dissecting, 479, 481.
- " electrolysis, 486.

Aneurism—

- „ embolism, 481.
- „ false, 481.
- „ heart, 419, 424, 425, 429.
- „ miliary, 869, 888.
- „ organisation, 482.
- „ popliteal, 481.
- „ phthisis chronica, 573.
- „ true, 481.
- „ thrombosis, 482.
- „ venesection, 485.
- „ verminous in horse, 265.
- Aneurism of thoracic aorta, 481-485.
- „ cough, brassy, 483.
- „ difference between radial pulses, 484.
- „ diastolic shock sound, 485.
- „ dysphagia, 483.
- „ dyspnoea, 482.
- „ „ comparison with asthma, 516.
- „ erosion, 482.
- „ expansive pulsation, 484.
- „ hæmoptysis, 483.
- „ murmurs, 485.
- „ paralysis, 482.
- „ thrill, systolic, 484.
- „ tracheal tugging, 484.
- „ Tuffnell's dietary, 485.
- „ voice, 483.
- Angina, 617.
- „ Ludovici, 615.
- Angina pectoris, 467.
- „ aortic incompetence, 453, 468.
- „ coronary arteries sclerosis, 468.
- „ death, sudden, 469.
- „ nitrites, 470.
- „ paroxysms, 468.
- „ table (Huchard's) of false and true, 469.
- Anginoid pains in fatty degeneration of heart, 418.
- Angio-cholitis, 738.
- „ suppurative, 742.
- Angio-neurotic hydrocephalus, 858.
- Angio-neurotic œdema, 995.
- „ colic, 996.
- „ heredity, 996.
- „ œdema glottidis, 996.
- Ankle clonus, 840.
- „ hemiplegia, 875.
- „ lateral sclerosis, 926.
- Ankylosis of vertebræ in arthritis deformans, 310.

Ankylostoma, duodenal, 264.

- Ankylostomiasis, 264.
- „ anaemia, 265.
- „ brickmakers, 265.
- „ colic, 265.
- „ diarrhoea, 265.
- „ dyspnoea, 265.
- „ eosinophilia, 347.
- „ fever, 265.
- „ miners, 265.
- „ œdema, 265.
- Anopheles mosquito, 241.
- Anorexia, 670.
- „ nervosa, 988.
- Anosmia, 1028.
- Anterior cranial nerve lesions, 1026.
- Anthraxis, 35.
- „ lungs, 556.
- „ tuberculosis, 556.
- Anthrax, 222.
- „ animals, 222, 223.
- „ bacillus, 222.
- „ bronchitis, 224.
- „ delirium, 224.
- „ diarrhoea, 224.
- „ fever, 224.
- „ hæmorrhagic lesions, 224.
- „ infective methods, 222, 223.
- „ intestinal form, 224.
- „ lymphatic glands, 223.
- „ man, 222.
- „ meningitis, 853.
- „ œdema of larynx, 224.
- „ pulmonary form, 224.
- „ spleen, 222.
- „ sporulation, 222.
- „ sputum, 224.
- „ toxæmia, 223, 224.
- Anthropophobia, 992.
- Antipeptones, 629.
- Antitoxin of diphtheria, 101.
- „ tetanus, 109.
- Antivenom, 282.
- Anuria, 780.
- „ catheter, 780.
- „ diphtheria, 780.
- „ lead poisoning, 780.
- „ obstructive, 780.
- „ non-obstructive, 780.
- „ phosphorus poisoning, 780.
- „ yellow fever, 203, 780.
- Aorta aneurism, 481.
- Aortic incompetence, 451.
- „ alcohol, 451.
- „ angina, 453, 468.
- „ apex beat, 452.
- „ death, sudden, 453.

- Aortic incompetence—
 " dyspnœa, 453.
 " heart, size of, 452.
 " murmur, diastolic aortic, 452.
 " Flint's, 453.
 " systolic mitral, 453.
 " pulsation, capillary, 453.
 " pulse, Corrigan's or water-hammer, 453.
 " rupture of valve, 453.
 " strain, 451.
 " syncope, 453.
 " syphilis, 451.
 Aortic second sound accentuated in granular kidney, 815.
 Aortic stenosis, 454.
 " apex beat, 455.
 " death, sudden, 455.
 " murmurs, diastolic aortic and mitral, 455, and systolic aortic, 455.
 " pulse, 455.
 " relative, 454.
 " thrill, 455.
 Aortic valves, incompetence of, 451.
 " rupture, 451, 453.
 " stenosis, 454.
 Aphasia, 957.
 " agraphia, 959.
 " amnesia verbalis, 961.
 " anarthria, 960.
 " aphemia, 950.
 " apraxia, 962.
 " auditory centre, 958, 961.
 " blindness (word and mind), 961.
 " deafness (word), 961.
 " " (mind), 962.
 " dysarthria, 961.
 " general paralysis of insane, 947.
 " glosso-kinæsthetic area, 958.
 " lead-poisoning, 290.
 " motor, 959.
 " motor centre, 958.
 " paraphasia, 960.
 " sensory, 961.
 " subcortical motor, 960.
 " visual centre, 958, 961.
 " writing centre, 959.
 Aphenia, 960.
 Aphonia, hysteria, 958.
 " laryngitis, 496, 500.
 Aphthous stomatitis, 609.
 Apoplectic stroke, 867.
 Apoplectiform bulbar paralysis, 918.
 Apoplexy, 867.
 " arterio-sclerosis, 477.
 " cerebral, 867.
 " ingravescent, 871.
 " kidney disease, 776, 814.
 " leukæmia, 368.
 " meningeal, 879.
 " pulmonary, 530, 531.
 Appendicitis, 687.
 " abscess, 689, 691.
 " age, 687.
 " bacteria, 688.
 " bladder, irritability, 691.
 " catarrhal, 688.
 " colic, 688, 704.
 " constipation, 691.
 " cysts, 690.
 " fecal concretions, 687, 689.
 " fever, 691.
 " fulminant, 692.
 " gangrenous, 689.
 " improvement, 691.
 " leucocytosis, 692.
 " M'Burney's point, 691.
 " obliteration of lumen, 690.
 " opium, 693.
 " pain, 690, 691.
 " patient, posture, 691.
 " perinephric abscess, 828.
 " peritonitis, 689, 692, 758.
 " pulse, 691, 692.
 " pus, signs, 692.
 " pylophlebitis, 690.
 " subphrenic abscess, 690.
 " suppurative, 689.
 " symptoms, absence, 693.
 " tenderness, 691.
 " tumor, 691, 692.
 " ulcerative, 689.
 Appendicular colic, 688, 704.
 Apraxia, 962.
 Aran-Duchenne type of muscular atrophy, 914.
 Argyll Robertson pupil, 108.
 " brain tumor, 956.
 " general paralysis of insane, 947, 1038.
 " light reflex arc, lesions, 1038.
 " tabes dorsalis, 934, 1038.
 Argylia, local and general, 35, 210.
 Arrhythmia, 463.
 Arsenical poisoning, 288.
 " anæmia, 288.
 " jaundice, 741.

- Arteries, calcification, 474.
 „ degenerations, 474.
 „ hypoplasia, 473.
 „ inflammation, 475.
 „ necrosis, 474.
 Arteriometer, 473.
 Arterio-sclerosis, 477.
 „ aneurism, 481.
 „ apoplexy, 477.
 „ cardiac hypertrophy, 425.
 „ 477.
 „ diabetes mellitus, 335.
 „ meningitis, 853.
 „ nephritis (interstitial and cardiac), 477, 809, 813.
 „ potassium iodide, 478.
 Arteritis, acute, 476.
 „ chronic, 476.
 „ heredity, 475.
 „ sclerotic form, 477.
 „ syphilitic, 476.
 „ thrombo-proliferous, 476.
 „ tuberculosis, 476.
 „ typhoid fever, 476.
 Arthritis—
 „ deformans, 307.
 „ dengue, 206.
 „ endocarditis, malignant, 440.
 „ gout, 320, 321.
 „ gonorrhoea, 305.
 „ hysteria, 988.
 „ influenza, 91.
 „ Malta fever, 88.
 „ pneumonia, 540.
 „ pyæmia, 138.
 „ rheumatic fever, 295.
 „ rheumatoid, 307.
 „ tabes dorsalis, 931, 935.
 Arthritis deformans, 307.
 „ anæmia, 311.
 „ ankylosis, 310.
 „ arthritis, 310.
 „ bacillus, 308.
 „ cartilage, 309, 310.
 „ children, 310.
 „ dislocation, 309.
 „ gout, 308.
 „ Heberden's nodes, 310.
 „ lymphatic glands, 310, 311.
 „ microbic theory, 308.
 „ muscular atrophy, 311.
 „ neural theory, 308.
 „ rheumatism, 308.
 „ spleen, 310.
 „ sweating of hands and feet, 311.
 Arthritis deformans—
 „ tremor, 310.
 Arthropoda, 272.
 Articular rheumatism, chronic, 301.
 Ascariasis, 261.
 Ascaris lumbricoides, 261.
 „ angio-cholitis, suppurative 742.
 „ asphyxia, 262.
 „ convulsions, 262.
 „ jaundice, 262.
 „ intestinal obstruction, 262.
 Ascending paralysis, acute, 910.
 Ascites, 4, 755.
 „ aspiration, 757.
 „ bladder distension, 757.
 „ cardiac valvular disease, 448.
 „ chylous, 270, 756.
 „ encysted, 756.
 „ fluid, 721, 755, 756.
 „ liver cirrhosis, 721.
 „ „ congestion, 708.
 „ navel prominence, 755, 756.
 „ chronic peritonitis, 769.
 „ peritoneum cancer, 770.
 „ toxæmia, 718, 755.
 „ tumors, 755.
 Asphyxia, local, 997.
 „ rabies, 229.
 Aspiration pneumonia, 547.
 „ lung abscess, 547.
 Astasia, 989.
 Asthenia, in Addison's disease, 404.
 „ cancer of liver, 729.
 „ „ œsophagus, 628.
 „ „ stomach, 666.
 „ cirrhosis of liver, 722.
 „ exophthalmic goitre, 399.
 „ leukaemia, 369.
 „ loss of thymus gland, 406.
 „ lymphadenoma, 378.
 „ sleeping sickness, 253.
 „ small-pox, 155.
 „ typhoid fever, 69.
 „ typhus fever, 198.
 Asthma, 514.
 „ age, 516.
 „ bronchiolitis, 515.
 „ bronchitis, eosinophilous, 515.
 „ Curschmann's spirals, 515.
 „ death, sudden, 516.
 „ diaphragm spasm, 514.
 „ diuresis, 515.
 „ dyspnoea, 516.

Asthma—

- „ hay, 491.
- „ nasal polypi, 514.
- „ posture, 516.
- „ sex, 514.
- „ sputum. Charcot's crystals and eosinophilous leucocytes, 515.
- „ thymic, 407.

Ataxia, cerebellar, 943.

- „ Friedreich's, 941.
- „ hereditary, 941.
- „ locomotor, 929.
- „ paraplegic, 940.

Ataxic paraplegia, 940.

- „ age, 940.
- „ Babinski's sign, 940.
- „ onset, 940.
- „ Romberg's sign, 940.
- „ sclerosis, 940.
- „ spasticity, 941.

Atelectasis, pulmonary, 527.

Atheroma, 478.

- „ age, 478.
- „ aneurism, 479, 481.
- „ aorta, 479.
- „ arteries (medium-sized), 480.
- „ calcification, 474, 479.
- „ pulmonary artery, 488.

Atheromatous abscess, 479.

- „ ulcer, 479.

Athetosis, 887.

- „ bilateral, 925.
- „ brain tumor, 955.

Athlete's heart, 462.

Atmospheric pressure, effects, 882.

Atony of intestines, 678, 682, 701.

Atrophy, 22.

- „ brain, 858.
- „ central, 913.
- „ cyanotic, of liver, 4, 707.
- „ degenerative, 22.
- „ gastric mucosa, 643.
- „ intestine, 678.
- „ leprosy, 125.
- „ limbs, 908.
- „ liver, 714, 715.
- „ muscular (progressive), 913.
- „ myocardial, 421.
- „ neuro-muscular, 839, 920.
- „ pigmentary, 22.
- „ simple, 22.
- „ spinal cord, 863.
- „ thyroid in myxœdema, 394.
- „ unilateral of face, 1001.
- „ yellow of liver, 724.

Attitude, paralysis agitans, 954.

- „ pseudo-hypertrophic paralysis, 922.

Auditory centres, 961.

- „ nerves, 1054.
- „ vertigo, 1057.

Aura in epilepsy, 971.

Aurium tinnitus, 1055.

Autochthonous pigmentation, 33, 35.

Autogenetic poisons, 274.

Autumnal catarrh, 491.

Axis-cylinder process, 832.

Axon, 832.

Babinski's sign, 875.

- „ ataxic paraplegia, 940.
- „ Brown-Sequard's paralysis, 844.
- „ cerebral hæmorrhage, 875.
- „ primary spastic paraplegia, 926.
- „ syringomyelia, 863.

Baby limbs in acute poliomyelitis, 908.

Bacillus, acid fast, 112.

- „ anthrax, 222.
- „ atrophy, acute yellow of liver, 724.
- „ botulinus, 279.
- „ cholera, 80.
- „ coli communis, 208.
- „ „ appendicitis, 688.
- „ „ gall stones, 730.
- „ „ liver abscess, 711.
- „ „ meningitis, 853.
- „ „ acute pancreatitis, 749.
- „ comma of Koch, 80.
- „ diphtheria, 93.
- „ diphtheroid in general paralysis of insane, 946.
- „ dysentery, 208.
- „ Eberth's, 59.
- „ enteriditis, 279.
- „ glanders, 219.
- „ Gartner, 279.
- „ Hoffmann, 612.
- „ ictroides, 202.
- „ influenza, 89.
- „ „ otitis media, 91.
- „ „ pneumonia, 559.
- „ Klebs-Löffler, 93.
- „ Koch's comma, 80.
- „ lactis aerogenes in pneumothorax, 596.
- „ lanceolatus, 535.
- „ leprosy, 123.

Bacillus—

- „ Limmitzer, 512.
- „ Lüffler, 93.
- „ mallei, 219.
- „ plague, 214.
- „ pneumonia, 535.
- „ pseudo-diphtheritic, 91, 612.
- „ pyocyaneus, 759.
- „ rheumatic fever, 291.
- „ rheumatoid arthritis, 308.
- „ smegma, 112.
- „ tetanus, 103.
- „ Timothy, fodder grasses, 112.
- „ tuberculosis, 109, 112.
- „ „ cerebro-spinal fluid, 851.
- „ „ endocarditis, 435.
- „ „ peritonitis, 759.
- „ „ pleural effusions, 601.
- „ „ sputum, 120, 563, 569, 577.
- „ „ urine, 800, 819.
- „ typhosus, in endocarditis, 433.
- „ „ gall-stones, 730.
- „ „ liver abscess, 711.
- „ „ pharyngeal ulcers, 619.
- „ „ pneumonia, 559.
- „ „ typhoid fever, 59.
- „ van Ermengem, 279.
- „ yellow fever, 202.

Bacteriological tests, 68, 98.

Bactericidal action of liver, 709, 714.

β -amido-butyric acid, 334.

Banti's disease, 371.

Barlow's disease, 387.

Barrel-shaped chest in emphysema, 520.

Basedow's disease, 396.

Bath treatment of fever, 48.

Beading of ribs in rickets, 315.

Bedsore in cerebral hæmorrhage, 877.

„ general paralysis of insane, 947.

„ multiple neuritis, 1017.

„ myelitis, 902.

„ syringomyelia, 864.

„ typhoid fever, 197.

Beer poisoning by arsenic, 288.

Bell's paralysis, 1050.

Bellyache, 704.

Bence Jones's disease, 789.

Beri-beri, 211.

„ cardiac failure, 213.

„ dropsy, 212.

„ knee-jerk, absence of, 203.

„ muscular paralysis, 212.

„ neuritis, 212, 1015.

„ reaction of degeneration, 212, 213.

„ respiratory failure, 212, 213.

Bile ducts, ascariides, 262.

„ cancer, 743, 745.

„ gall stones, 732, 733, 734.

„ inflammation, 738.

„ suppurative, 742.

„ obstruction, 733, 745.

„ occlusion of, in syphilis, 34, 741.

„ stenosis, 745.

Bile in urine, 793.

Bile pigmentation, 34.

Bilharzia hæmatobium, 254.

„ diarrhoea, 255.

„ hæmaturia, 255, 790.

Biliary abscess, liver, 711, 743.

Biliary cancer, liver, 728.

Biliary cirrhosis, liver, 714, 715.

Biliary colic, 734.

Biliousness, 710, 738.

Bilious diarrhoea, 681.

Bilious remittent fever, 244.

„ „ vomiting, 244.

Bilious vomiting, 247, 726.

Bilirubin, 34.

Bimanual examination of kidney, 772.

Birth palsies, 924.

Bitemporal neuralgia in syphilis, 186.

Black vomit, 202.

Blackwater fever, 246.

Bladderworm, 257.

Bladder, ova of bilharzia, 255.

„ paralysis, in myelitis, 902.

„ tuberculosis, 818.

„ typhoid fever, 71.

“Bleeders,” 380.

Blepharospasm, 1054.

Blindness, hysteria, 988.

„ lead poisoning, 290.

„ leprosy, 125, 127.

„ night, scurvy, 385.

„ small-pox, 153.

„ vide also amaurosis.

Blood, corpuscles, 342.

„ examination, 341, 342.

„ destruction, 343.

„ diseases, 341.

Blood—

- „ formation, 348.
- „ pigmentation, 33, 34.
- „ plates, 18, 347.
- „ stains, 342.
- „ stagnation, 8.
- Blood, characters of, in ankylostomiasis, 265.
- „ appendicitis, 692.
- „ chlorosis, 350.
- „ chloroma, 372.
- „ cholera, 82.
- „ diabetes mellitus, 329, 330.
- „ gout, 319.
- „ hæmoptysis and hæmatemesis, 13, 14.
- „ Hodgkin's disease, 377.
- „ leukæmia, 361, 363.
- „ paroxysmal hæmoglobinuria, 389.
- „ pernicious anæmia, 354.
- „ pneumonia, 537.
- „ poisons, 388.
- „ rheumatic fever, 295.
- „ scurvy, 385.
- „ septicæmia, 135.
- „ secondary anæmia, 358.
- „ splenic anæmia, 370.
- „ stomach cancer, 665, 667.
- „ trichinosis, 267.
- „ typhoid fever, 60.
- „ tuberculosis, 119.
- „ urine, 790.
- Blood letting, in arterio-sclerosis, 478.
- „ cerebral hæmorrhage, 877.
- „ emphysema, 522.
- „ heart disease, 459.
- „ sunstroke, 293.
- Blood-serum therapy, in diphtheria, 101.
- „ plague, 218.
- „ tetanus, 109.
- Blue-black line on gums in lead poisoning, 290.
- Boiled sago stools in dysentery, 209.
- Boils, in diabetes mellitus, 335.
- Bones, changes in, achondroplasia, 1003.
- „ acromegaly, 1001.
- „ osteomalacia, 1003.
- „ osteitis deformans, 1002.
- „ pulmonary osteo - arthropathy, 1002.
- „ rickets, 314.
- „ syphilis, 188.

- Bone marrow, in chloroma, 373.
- „ leukæmia, 364, 367, 369.
- „ lymphadenoma, 376.
- „ pernicious anæmia, 356.
- Borborygmi, 632.
- Bothriocephalus latus, 256.
- Bow legs in rickets, 316.
- Booming second cardiac sound in aneurism, 485.
- Bowel diseases, 672 (*vide* intestine)
- β -oxybutyric acid, 333.
- Brachial plexus, 1022.
- Bradycardia, 466.
- Brain, diseases of—
- „ abscess, 893.
- „ anæmia, 865.
- „ aneurism, 888.
- „ compression of, in meningeal hæmorrhage, 867.
- „ tubercular meningitis, 852.
- „ embolism, 884.
- „ fever, 193.
- „ glioma, 952.
- „ hæmorrhage, 867.
- „ inflammations, 890.
- „ cedema, 867, 892.
- „ paralysis in typhoid fever, 69.
- „ porencephaly, 887.
- „ railway, 994.
- „ sclerosis, 946.
- „ softening, 12, 884.
- „ thrombosis, 884.
- „ tubercle, 851, 952.
- „ tumors, 951.
- Brain abscess, 893.
- „ aphasia, 895.
- „ bacteria, 893.
- „ bronchiectasis, 513, 894.
- „ capsule, 894.
- „ contents, 894.
- „ ear, middle, 893.
- „ fever, 895.
- „ headache, 895.
- „ localisation, 895.
- „ leucocytosis, 895.
- „ optic neuritis, 895, 896.
- „ skull fracture, 894.
- „ sites, 894.
- „ symptoms absent, 895.
- „ typhoid fever, 894.
- „ vomiting, 895.
- Brain embolism and thrombosis, 884, 886.
- „ consciousness, 885.
- „ mode of onset, 885.
- Brain sclerosis, 913.

Brain sclerosis—

- „ encephalitis, 892.
- „ hemiplegia of children, 886.
- „ insular, 949.

Brain tumors, 951.

- „ aphasia, 954.
- „ athetosis, 955.
- „ chorea, 955.
- „ convulsions, 953. 956.
- „ death, sudden, 956.
- „ glycosuria, 956.
- „ giddiness, 953.
- „ headache, 952.
- „ hemianopsia, 954. 955.
- „ hemiplegia, 954.
- „ optic neuritis, 952.
- „ signal symptom, 953.
- „ vomiting, 952.

Brassy cough in aneurism of aorta, 483.

Breakbone fever, 205.

Breast pain, 467.

Breast, pigeon-shaped in rickets, 316.

- „ tonsils hypertrophy, 623.

Breathing, stridor in, abductor paralysis of larynx, 1061.

- „ laryngismus stridulus, 502.

Breath, odour of, in alcoholism and cerebral hæmorrhage, 876.

- „ diabetes mellitus, 333.

Brickred urates, 783.

Brickmakers' disease, 265.

Bright's disease, 798.

- „ acute, 799.
- „ chronic, 806 (*vide* nephritis).

British yellow fever, 204.

Broadbent's law, 873.

Broca's convulsion, 833, 959.

Bromides in epilepsy, 976.

Bromism, 977.

Bronchial asthma, 492.

Bronchial breathing, pneumonia, 539.

- „ phthisis, 580.

Bronchial casts in sputum, 510.

Bronchiectasis 511.

- „ acquired, 511.
- „ bacillus of Limmitzer, 512.
- „ brain abscess, 513, 894.
- „ bronchorrhœa, 512.
- „ cavities, 512.
- „ children in acute miliary tuberculosis, 512.
- „ congenital, 511.
- „ cough, 512.

Bronchiectasis—

- „ cylindrical, 511.
- „ Dittrich's plugs, 513.
- „ emphysema, 512.
- „ hæmoptysis (rare), 513.
- „ phthisis, chronic, 573.
- „ pneumonia, interstitial, 512.
- „ saccular, 511.
- „ sputum, 513.
- „ Traube's plugs, 513.
- „ tracheal injections, 513.

Bronchiolitis, 515.

Bronchitis, acute, 503.

- „ bacteria, 503.
- „ capillary, 543.
- „ chronic, 507.
- „ dyspnœa, 505.
- „ kidney, granular con-
tracted, 814.
- „ measles, 173, 505.
- „ pain, 505.
- „ pneumonia, 505.
- „ pulse, 505.
- „ rhonchi, 505.
- „ sputum, 504, 505.
- „ tuberculosis (miliary), 505.
563.
- „ typhoid fever, 66, 74, 505.
558.
- „ whooping-cough, 179, 508.

Bronchitis, chronic, 507.

- „ bronchorrhœa, 508.
- „ dust inhalation, 507.
- „ eosinophilous, 515.
- „ fibrinous, 509.
- „ foetid, 508.
- „ gout, 507.
- „ heart disease, 446, 448, 507.
- „ kidney disease, 507.
- „ Nauheim treatment, 509.
- „ pituitous catarrh, 508.
- „ plastic, 509.
- „ putrid, 508.

Bronchocele, 392.

Bronchophony, in phthisis, 579.

- „ pneumonia, 539.

Broncho-pneumonia, 543 (*see* lobular pneumonia).

Bronchorrhœa, in abscess of lung,

- 550, 551.
- „ bronchiectasis, 512.
- „ chronic bronchitis, 508.

Bronchus foreign body, 517, 552.

- „ abscess, 517.
- „ hæmorrhage, 517.
- „ Bronchus obstruction, 517.
- „ dyspnœa, 517.

- Bronchus obstruction—
 „ gangrene of lung, 552.
 Bronzing of skin in diabetes of
 Hanot, 330.
 Brown atrophy of myocardium, 421.
 Brown induration of lung, 4, 526.
 Brown-Sequard's paralysis, 843.
 Brown skin in Addison's disease,
 405.
 Bruit de diable in chlorosis, 351
 Bubo in plague, 215.
 Bubonic plague, 216.
 Bulbar paralysis, 917.
 „ acute, 918.
 „ aphonia, 919
 „ apoplectiform, 918
 „ cerebellar gait, 918.
 „ chronic, 918.
 „ nystagmus, 918.
 „ pseudo, 919, 950
 Bulimia, 670.
 Burns, duodenal ulcers, 660
 Button hole mitral, 444.
 Cachexia in gastric cancer, 666.
 „ lead poisoning, 291
 "Cadaveric" position of vocal cords,
 105c.
 Caisson disease, 882.
 „ coma, 883.
 „ fissures in cord, 883
 „ gas emboli, 883
 „ pain, 883
 „ paralysis, 883
 Calcareous infiltration, 33
 „ arteries, 474
 „ heart valves, 443.
 „ trichiniasis, 266.
 „ tubercular foci, 572.
 Calculi, biliary, 731.
 „ pancreatic, 754.
 „ renal, 797.
 „ urinary, 795.
 Calculus pyelitis, 820.
 Calf lymph, 159.
 „ glycerinated, 159
 Camp fever, 192
 Cancer, of bile ducts and gall
 bladder, 743.
 „ brain, 952.
 „ intestine, 698.
 „ kidney, 82.
 „ larynx, 502.
 „ liver, 728.
 „ lung, 591.
 „ myocardium, 431.
 „ œsophagus, 627
 Cancer—
 „ pancreas, 752.
 „ peritoneum, 769.
 „ pleura, 607.
 „ stomach, 661.
 „ suprarenals, 406.
 „ thymus, 407.
 „ thyroid, 400.
 Cancer bodies, 249.
 Cancrum oris, 612.
 Capillary pulsation in aortic incom-
 petence, 453.
 Capillary bronchitis, 543.
 Capsule, internal 835.
 Caput medusæ, 718.
 Carbuncle, diabetes mellitus, 335
 Carcinoma, 38 (*see* cancer).
 Cardiac cases of malignant endocar-
 ditis, 439
 Cardiac murmurs, anæmic, 351.
 „ aortic stenosis, 455.
 „ chlorosis, 351.
 „ pernicious anæmia, 356.
 Cardiac murmurs, organic, in aortic
 incompetence, 452.
 „ aortic stenosis, 455.
 „ endocarditis (acute) 437.
 „ mitral incompetence, 447.
 „ mitral stenosis, 450.
 „ pulmonary incompetence
 and stenosis 457.
 „ tricuspid incompetence, 456.
 „ tricuspid stenosis, 457.
 Cardiac neuroses, 461.
 Cardiac overstrain, 452.
 Cardiac septa, defects, 408.
 Cardiac tonics, heart disease, 459.
 „ influenza, 92
 Cardiac vertigo, 1057
 Carnes, vertebral, 617, 809
 Carpo-pedal spasms, 980
 Cascara sagrada, constipation, 703
 Caseation, 21.
 „ tubercle, 114.
 Caseous broncho-pneumonia tuber-
 culosis, 554.
 Casts, bronchi in asthma, 515.
 „ fibrinous bronchitis, 510.
 „ colon in mucous colitis, 684.
 „ urinary tubules, 793.
 Casts, renal, 793.
 „ blood-cell, 795
 „ colloid, 794
 „ epithelial, 794.
 „ fatty, 794.
 „ fibrin, 794
 „ granular, 795

Casts—

„ hyaline, 794.

„ waxy, 794.

Casts, tube, acute Bright's disease, 803.

„ chronic parenchymatous nephritis, 808.

„ chronic interstitial nephritis, 815.

„ health, 788.

„ passive congestion of kidney, 777.

„ waxy kidney, 776.

Catalepsy, hysteria, 987, 989.

Cataract, diabetic, 335.

Catarrh, autumnal, 491.

„ bronchial, 503, 506.

„ gastric, 638, 641.

„ nasal, 489.

Catarrhal appendicitis, 688.

„ enteritis, 683.

„ laryngitis, 132, 496.

„ pneumonia, 543.

„ stomatitis, 608.

Cauda equina in tubercular meningitis, 852.

Cavernous breathing, 580.

Cavities in lung, 573.

„ signs, 550, 580.

Cayenne pepper like deposit of uric acid, 784.

Cell inclusions in tumors, 40.

Cellulitis, cervical, 615.

„ orbital affecting ophthalmic branch of fifth nerve, 1044.

Central scotoma, 1033.

Cephalic tetanus, 107.

Cerebellar hereditary ataxia, 943.

Cerebellar lesions, 956.

Cerebral abscess, 893.

Cerebral arteries—

„ aneurism, 868, 888.

„ aneurisms, multiple, 859.

„ arterio-sclerosis, 477.

„ disease, 868.

„ embolism, 884.

„ thrombosis, 884.

Cerebral cortex, 838.

Cerebral diplegia, 924.

Cerebral hæmorrhage, 867.

„ age, 869.

„ aneurisms, miliary, 869.

„ apoplexy, 867.

„ apoplexy, ingravescent, 871.

„ apoplectic cyst, 870.

„ artery (Charcot's), 868.

Cerebral hæmorrhage—

„ athetotic movements, 875.

„ Babinski, positive, 875.

„ blood letting, 877, 878.

„ Cheyne Stoke's breathing, 871.

„ children, 869.

„ choreiform movements, 875.

„ coma, 876.

„ conjugate deviation, 872.

„ consciousness, 871.

„ convulsions, 872, 874.

„ degenerations secondary, 871.

„ emboli, 859.

„ hemiplegia, 873, 874.

„ hemiplegia flasque, 875.

„ monoplegia (cerebral), 873

„ paralysis, 872.

„ pulse, 872.

„ pupils, 872, 876.

„ purgation, 877.

„ reflexes, 872.

„ rigidity, early and late, 875.

„ sites, 868.

„ speech, blurring, 697.

Cerebral monoplegia, 873.

Cerebral sinuses and veins, thrombosis, 889.

Cerebral softening, 884.

Cerebro-spinal fever, 77 (*see* cerebro-spinal meningitis).

Cerebro-spinal fluid, in epidemic cerebro-spinal meningitis, 77, 79.

„ fracture of skull, 880.

„ sleeping sickness, 252.

„ tubercular meningitis, 853.

Cerebro-spinal meningitis, epidemic and sporadic, 77.

„ albuminuria, 78.

„ diarrhoea, 78.

„ delirium, 78.

„ eyeball inflammations, 78.

„ fever, 78.

„ geographical distribution, 77.

„ herpes of mouth, 78.

„ insomnia, 78.

„ lumbar puncture, 79.

„ micrococcus, 77.

„ opisthotonos and orthotonos, 78.

„ posterior basal, 77.

„ rashes, 78.

„ strabismus, 78.

„ tetanic spasms, 78.

- Cervical glands, in diphtheria, 97.
 „ scarlet fever, 167.
 Cervical pachymeningitis hypertrophica, 847.
 Cervical nerve-roots, lesions, 1020.
 Cestodes, 255.
 Chalicosis, 556.
 Chancre, 184.
 Charbon, 222.
 Charcot's artery of cerebral hæmorrhage, 863.
 Charcot's crystals in asthma, 515.
 Charcot's joint, 931, 935.
 Chaulmoogra oil in leprosy, 128.
 Cheek, gangrene, 612.
 Chest deformity, in chronic pericarditis, 416.
 „ pleurisy, 602.
 „ rickets, 315.
 „ chronic tonsillitis, 623.
 Cheyne-Stokes breathing, in apoplexy, 872.
 „ uræmia, 783.
 Chicken breast in hypertrophy of tonsils, 623.
 Chicken-pox, 162.
 Child crowing, 502.
 Children, apoplexy, 869.
 „ cancrum oris, 612.
 „ colitis (mucous), 685.
 „ empyema, 601.
 „ enteritis, 681.
 „ fatty diarrhœa, 747.
 „ lavage in diarrhœa, 700.
 „ lobar pneumonia, 539.
 „ pericarditis, 413.
 „ rheumatic fever, 296.
 „ tuberculosis of intestine, 694.
 „ typhoid fever, 73.
 Chloral hydrate rash, 176.
 Chlorides diminished excretion of, gastric catarrh, 645.
 „ lobar pneumonia, 537, 538.
 Chloroma, 372.
 Chlorosis, 349.
 „ amenorrhœa, 351.
 „ anæmia, 350, 351.
 „ bruit de diable, 351.
 „ cardiac murmurs, 351.
 „ colour index, 350.
 „ constipation, 350, 351.
 „ dyspnœa, 350, 351.
 „ fever, 46.
 „ gastric ulcer, 351, 654.
 „ giddiness, 351, 352.
 „ hæmoglobin, 350.
 Chlorosis—
 „ hæmogenesis, defective, 350, 354.
 „ hypoplasia, 349.
 „ iron, 350, 352.
 „ normoblasts, 350.
 „ œdema, 350, 351.
 „ poikilocytes, 350.
 „ sex, 349.
 „ thrombosis, 351, 389.
 Choked disc, hydrocephalus, 859, 860.
 Cholæmia, 739.
 Cholangitis, 738.
 „ large ducts, 738.
 „ small ducts, 739.
 Cholecystitis, 732, 737.
 Cholecystotomy, 736.
 Cholera, 80.
 „ albuminuria, 85.
 „ anuria, 83.
 „ bacillus, 80.
 „ blood, 82.
 „ collapse, 83.
 „ cramps, 82.
 „ cyanosis, 82.
 „ European, 84.
 „ facies, 83.
 „ geographical distribution, 80.
 „ Haffkine's vaccine, 85.
 „ intravenous injection, 86.
 „ mushroom poisoning, resemblance, 85, 279.
 „ nostras, 84.
 „ nephritis, 799.
 „ premonitory stage, 82.
 „ ptomaine poisoning, resemblance, 85, 279.
 „ rash, 84.
 „ reaction, 84.
 „ sicca, 82.
 „ stools, 82, 83.
 Cholera infantum, 681, 685.
 Cholesteatoma, 857.
 Cholesterin, gall stones, 731.
 Choluria, 793.
 Chondroma, 38.
 „ lung, 591.
 Choreæ, 965.
 „ acute, 965.
 „ age, 965.
 „ brain tumor, 955.
 „ chronic, 968.
 „ endocarditis, 434, 965.
 „ gravidarum, 968.
 „ hemichorea, 966.

Chorea—

- „ Huntingdon's, 968.
- „ hysterical, 959.
- „ major, 969.
- „ maniacal, 967.
- „ movements, 967.
- „ pregnancy, 955.
- „ rheumatism, 297, 955.
- „ rhythmic, 969.
- „ senile, 959.
- „ sex, 965.
- „ spastic, 925.
- „ speech, 956.
- „ Sydenham's, 955.
- Chorea spastica, 925.
- Chronic deforming gout, 320.
- Choroid miliary tubercles, 563.
- Chvostek's symptom in tetany, 981.
- Chylous ascites, 270.
- „ hydrocele, 271.
- Chylothorax, 596.
- Chyluria, 270, 790.
- Cinchonism, 299.
- Circumflex nerve paralysis, 1024.
- Cirrhosis, kidney, 809.
- „ liver, 713.
- „ liver with pigmentation, 752.
- „ liver cancer, 728.
- „ stomach, 643.
- Clanging cough in aneurism of aorta, 483.
- Clasp knife action in primary spastic paralysis, 925.
- Claustrophobia, 992.
- Claw-like hand (main en griffe), in progressive muscular atrophy, 916.
- „ pachymeningitis, 847.
- „ ulnar nerve paralysis, 1025.
- Clay-coloured stools, in chronic gastric catarrh, 641.
- „ obstruction of bile ducts, 741.
- Climatic influence in asthma, 514.
- „ chronic interstitial nephritis, 817.
- „ tuberculosis, 583.
- Clownism in hysteria, 987.
- Club-foot in cerebellar ataxia, 943.
- „ pseudo-hypertrophic paralysis, 923.
- Coagulation necrosis, 20.
- Coal miners' lung disease, 555.
- Cocainism, 285.
- Coccidia, in cancers, 249.
- „ cattle, 249.
- „ molluscum contagiosum, 249.

Coccidia—

- „ Paget's disease of nipple, 249.
- „ pheasants, 249.
- „ pigs, 249.
- „ rabbits, 248.
- „ some skin diseases, 249.
- „ tumors, 39.
- Coccidium oviforme, 248.
- Cochlear nerve, 1054.
- Codliver oil in phthisis, 585.
- Cœliac plexus in pancreatic disease, 747.
- Cogwheel like inspirations, 579.
- Coffee-ground vomit, 657, 665.
- Cohnheim's end arteries, 11.
- „ theory of tumor formation, 39.
- Cold in head, 488.
- Cold pack treatment of fever, 48.
- Colic, appendicular, 688, 704.
- „ biliary, 706, 733, 734.
- „ gastric, 735.
- „ hepatic, 706, 733.
- „ intestinal, 704.
- „ lead, 735.
- „ renal, 706, 733.
- Colitis, bilharzial, 255.
- „ mucous, 684, 685.
- „ ulcerative, 684.
- Collapse of lung, acquired, 523.
- „ bronchial pressure, 552.
- „ congenital, 522.
- „ cyanosis, 523, 524.
- „ dyspnoea, 524.
- „ pleurisy, 601.
- „ pneumonia, lobular, 544.
- „ 545.
- „ rickets, 524.
- „ whooping-cough, 179, 524.
- Colles's law, 189.
- Colliquative necrosis, 21.
- Colloid cancer, of peritoneum, 759.
- „ rectum, 698.
- „ stomach, 662.
- Colloid substance in cancer, 28.
- „ thyroid gland, 28, 390.
- Color index, 341.
- „ chlorosis, 350.
- „ leukæmia, 361.
- „ lymphadenoma, 377.
- „ pernicious anæmia, 354.
- „ secondary anæmias, 358.
- Coma, in alcoholism (acute), 274, 876.
- „ apoplexy, 876.
- „ cirrhosis of liver, 720, 722.

- Coma—
 „ diabetes mellitus, 333, 334, 876.
 „ food poisoning, 279.
 „ meningitis (tubercular), 852.
 „ opium poisoning, 283, 876.
 „ phosphorus poisoning, 287
 „ pneumonia, 539.
 „ scarlet fever, 168.
 „ sleeping sickness, 253.
 „ small-pox, 152.
 „ sunstroke, 292.
 „ typhoid fever, 64.
 „ typhus fever, 197.
 „ uræmia, 782, 87c.
 „ yellow atrophy, liver, 725.
 „ yellow fever, 203.
 Coma vigil, 64.
 Combined valvular lesions, 458.
 Common bile-duct obstruction, 733.
 Compensation in cardiac disease,
 445.
 „ disturbances, 446.
 „ failure, 447, 459.
 Compensatory emphysema, 520.
 Compressed air, disease, 882.
 Compression myelitis, 899.
 „ abscess, 900.
 „ anæsthesia dolorosa, 900.
 „ cancer, 900.
 „ laminectomy, 903.
 „ paraplegia, 900.
 „ pupils, dilated, 900.
 „ spasticity, 900.
 „ tubercular, 901.
 Concretions, fæcal, 687.
 Condylomata in syphilis, 185.
 Cone-shaped heart, 426.
 Congenital syphilis, 183.
 „ valvular disease, 456, 457.
 Congestion of lungs, 446, 525.
 „ hypostatic, 526.
 „ pigmented cells in sputum,
 526.
 Conjugate deviation, 1042.
 „ apoplexy, 872.
 Conjunctivitis in gonorrhœa, 505.
 Constipation, 701.
 Consumption (galloping), 564 (*see*
 tuberculosis).
 „ pulmonary, 561.
 Continual cough, 178.
 Contracted kidneys, 809.
 Contractures, in hemiplegia, 875.
 „ hysteria, 988.
 „ progressive muscular
 atrophy, 916.
 Convalescence precaution, typhoid
 fever, 71.
 Convulsions in ascariasis, 262.
 „ brain tumors, 953.
 „ cerebral hæmorrhage, 872.
 „ dentition, 977.
 „ epilepsy, 972.
 „ food poisoning, 279.
 „ general paralysis, 947.
 „ hemiplegia in children, 887.
 „ hydrophobia, 228.
 „ hydrocephalus, 860, 861.
 „ hysteria, 987.
 „ infantile, 977.
 „ lead poisoning, 290.
 „ measles, 174, 978.
 „ meningitis (tubercular), 852.
 „ nephritis, acute, 803.
 „ poliomyelitis, acute, 907.
 „ pneumonia, 977.
 „ rickets, 317, 977.
 „ scarlet fever, 166, 168, 977.
 „ small-pox, 150, 153.
 „ tetanus, 107.
 „ typhus fever, 197.
 „ uræmia, 783.
 „ whooping-cough, 179, 977.
 „ yellow fever, 203.
 Convulsions, Jacksonian, 838.
 „ brain tumor, 954.
 „ epilepsy, 971, 974.
 „ cortical lesions of seventh
 nerve, 1052.
 „ meningitis, 852.
 „ uræmia, 783.
 Co-ordination disturbances, in
 chorea, 967.
 „ tabes dorsalis, 932, 933.
 Copaiba rash, 176.
 Copper test for sugar, 331.
 Coprolalia, 970.
 Corneal ulcer, in exophthalmic
 goitre, 398.
 „ paralysis of sensory portion
 of fifth nerve, 1045.
 „ small-pox, 154.
 Coronary arteries, in angina pec-
 toris, 467.
 „ arterio-sclerosis, 424, 467.
 „ embolism, 424.
 „ stenosis, 417, 419, 423, 424.
 „ thrombosis, 424.
 Corpora, amylacea, 30.
 „ lutea, 35.
 Corpora quadrigemina lesions, 955.
 Corrigan's pulse, 453.
 Coryza, acute, 488.

Coryza—

- „ measles, 173.
- Cough, in adenoids (barking), 495.
- „ bronchitis, acute, 504.
- „ chronic, 507.
- „ bronchiectasis (spasmodic), 512.
- „ croupous, 102.
- „ diphtheria, 102.
- „ emphysema, 518, 520.
- „ hysteria (barking), 989.
- „ laryngitis, 499.
- „ paroxysmal, 178, 512, 550.
- „ persistent, 178.
- „ pertussis, 178.
- „ phthisis, 578.
- „ pleurisy, 602.
- „ pneumonia, 538, 550.
- „ relapsing fever, 53.
- „ "stomach," 495.
- „ winter, 507.
- Cows and scarlet fever, 163.
- Cow's milk as cause of enteritis, 681.
- Cow-pox, 159.
- Cracked pot sound in lung cavity, 580.
- Cramp, telegraphist's, 978.
- „ writer's, 978.
- Cramps in muscles, in beri-beri, 212.
- „ cholera, 83.
- „ sciatica, 1007.
- „ uræmia, 782.
- Craniotabes in rickets, 315.
- Crepitations fine, in early phthisis, 579.
- Cretinism, 395.
- „ abdomen, 396.
- „ endemic, 395.
- „ epiphyses, 396.
- „ intelligence, 396.
- „ sporadic, 396.
- Cretinoid state, 396.
- Crises in tabes dorsalis, 935.
- Crisis in pneumonia, 538.
- Critical evacuations in fever, 44.
- Crossed hemiplegia, 874.
- „ paralysis, 955.
- Croup, 97.
- „ membranous, 102.
- „ spasmodic, 317, 502.
- Croupous enteritis, 683.
- „ pneumonia, 534.
- Crustaceans and guinea worm, 268.
- Crutch palsy, 1024.
- Crura cerebri, lesions, 955.
- Cry, epileptic, 972.
- „ hydrocephalic, 852.

Cry—

- „ hysterical, 986.
- Cryptogenetic endocarditis, 439.
- Cure in phthisis, 572.
- Curschmann's spirals, 515.
- Cutaneous nerve external, neuritis of, 1026.
- Cyanosis, 4.
- „ caseous tuberculosis (acute) of lung, 569.
- „ emphysema, 520.
- „ miliary tuberculosis of lung, 563.
- „ opium poisoning, 283.
- „ pneumonia, 541.
- „ valvular disease (congenital) of heart, 457.
- „ whooping-cough, 179.
- Cyanotic atrophy of liver, 4, 707.
- Cyan-methæmoglobin, 388.
- Cyclical albuminuria, 788.
- Cycloplegia, 1039.
- Cystic disease, of kidneys, 825.
- „ liver, 754, 825.
- „ pancreas, 754, 825.
- Cystic duct, obstruction of, 733.
- Cysticercus cellulosæ, 257.
- Cystin calculi, 796.
- Cystinuria, 787.
- Cysts, in brain (apoplectic), 870.
- „ (porencephalic), 887.
- „ kidneys, 813, 825.
- „ pancreas, 753.
- „ peritoneum, 753, 770.
- Cystitis in bilharzia hæmatobia, 255.
- „ gonorrhœa, 305.
- „ myelitis, 902.
- „ syringomyelia, 864.
- „ tabes dorsalis, 935.
- Cytological characters of pleural effusion, 600.
- Dancing chorea, 969.
- Dandy fever, 206.
- Deafness, adenoids, 495.
- „ brain tumor, 955.
- „ diphtheria, 98.
- „ hysteria, 988.
- „ Meniere's disease, 1057.
- „ mind, 962.
- „ chronic nasal catarrh, 491.
- „ scarlet fever, 168, 171.
- „ small-pox, 153.
- „ tabes dorsalis, 935.
- „ wax, 1055.
- „ words, 961.
- Death, sudden, in angina pectoris, 469.

Death, sudden—

- „ aneurism of aorta, 485.
- „ aortic incompetence, 453.
- „ aortic stenosis, 455.
- „ apoplexy, 877.
- „ asthma, 516.
- „ brain embolism and thrombosis, 885.
- „ brain tumour, 956.
- „ caisson disease, 883.
- „ cholera, 83.
- „ coronary artery occlusion, 424.
- „ diphtheria, 97.
- „ gastric ulcer, 656.
- „ hæmatomyelia, 882.
- „ hæmoptysis of aneurism, 483.
- „ „ phthisis, 577.
- „ heart, fatty degeneration, 418.
- „ heart, fatty infiltration, 419.
- „ hematemeses, 657.
- „ mitral incompetence, 448.
- „ nephritis (acute), 804.
- „ nephritis (chronic parenchymatous), 809.
- „ opium poisoning, 283.
- „ phthisis, 576.
- „ pleural effusion, 606.
- „ sunstroke, 292.
- „ thymic hypertrophy, 407.

Decubitus, dorsal, in typhoid fever, 64, 65.

Degeneration, reaction, 839.

Degeneration, ascending and descending, 863.

Deiter's nucleus and nystagmus, 1043.

Delayed sensation in tabes dorsalis, 933.

Delirium, in alcoholism, 276.

- „ cirrhosis, liver, 720, 722.
- „ cordis, 463.
- „ erysipelas, 141.
- „ expansive, 947.
- „ hydrophobia, 228.
- „ maniacal in chorea, 967.
- „ meningitis, tubercular, 852.
- „ opium poisoning, 284.
- „ pneumonia, 539.
- „ relapsing fever, 54.
- „ small-pox, 151, 156.
- „ tremens, 279.
- „ typhoid fever, 61, 64, 65, 73, 74.
- „ typhus fever, 195, 197.

Delirium—

- „ uræmia, 782.
- „ yellow atrophy, liver, 726.

Deltoid paralysis, 1026.

Delusions of grandeur, 947.

Dementia paralytica, 944.

Dendrites of nerve-cells, 832.

Dengue, 205.

- „ fever, 206.
- „ incubation period, 205.
- „ joints, 205.
- „ pains in joints, 206.
- „ relapses, 206.

Dentition, in diabetes mellitus, 333.

- „ hereditary syphilis, 189.
- „ rickets, 315.
- „ stomatitis, 608, 611.

Desquamation in measles, 173.

- „ measles (German), 177.
- „ scarlet fever, 167.

Dextrocardia, 408.

Diabète Bronze, 330.

Diabetes insipidus, 338.

- „ fourth ventricle, 338.
- „ health good, 339.
- „ heredity, 338.
- „ polyuria, 338.
- „ sex, 338.

Diabetes mellitus, 327.

- „ age, 333.
- „ air hunger, 334.
- „ albuminuria, 335.
- „ appetite, 333.
- „ bile duct cancer, 745.
- „ blood, 329.
- „ staining, 330.
- „ carbuncle, 335.
- „ cataract, 335.
- „ coma, 333, 334.
- „ diet, 336.
- „ fat emboli, 329.
- „ fat in urine, 790.
- „ gangrene, 335.
- „ „ lung, 552.
- „ hepatic theory, 328.
- „ heredity, 327.
- „ hyperglycæmia, 328, 329.
- „ Langerhan's islands, 747.
- „ liver cirrhosis, 330.
- „ lung diseases, 330.
- „ mania, 335.
- „ microbic theory, 329.
- „ neuritis, 335, 1015.
- „ nervous theory, 327.
- „ pancreatic theory, 329, 747, 752.
- „ phthisis, 335.

Diabetes mellitus—

- „ pneumonia, 330, 552.
- „ sclerosis of cord, 944.
- „ tests for sugar, 330, 337.
- „ thirst, 333.
- „ urine, 330, 332, 333.
- „ albumin, 332, 335.
- „ acetone, 333, 792.
- „ chloride excess, 330.
- „ diacetic acid, 333, 793.
- „ polyuria, 333.
- „ urea excess, 330, 781.
- „ uric acid excess, 784.

Diabetes, phosphatic, 785.

Diabetic albuminuria, 335.

- „ coma, 333, 334, 793.
- „ diet, 336.
- „ eczema, 335.
- „ phthisis, 335.
- „ pruritus pudendi, 335.

Diacetic acid, 333, 793.

Diaphragm, paralysis, 1021.

- „ spasm, 1021.

Diarrhœa, 698.

- „ bilious, 681.
- „ children, treatment, 700.
- „ cholera, 83.
- „ dysentery, 209.
- „ fatty, 681, 747 (children).
- „ gastric catarrh (acute) of children, 639.
- „ hepatic cirrhosis, 699.
- „ Hill, 684.
- „ hysteria, 988.
- „ lenteric, 685.
- „ obstruction (chronic) of bowel, 676.
- „ phthisis (waxy intestine), 678.
- „ summer, 685.
- „ tubular, 684.
- „ typhoid fever, 63.
- „ uræmia, 783.

Diastolic aortic murmur, 452.

- „ mitral murmur, 450, 455.

Diastolic thrill in aortic incompetence, 452.

Diathesis, uric acid 784.

Dicrotic pulse, in typhoid fever, 61.

Diet, aneurism, 485.

- „ constipation, 703.
- „ diabetes, 336.
- „ diarrhœa, 700.
- „ dysentery, 211.
- „ dyspepsia, 633.
- „ fever, 50.
- „ gastric ulcer, 659.
- „ gout, 333.

Diet—

- „ nephritis, acute, 805.
- „ „ chronic, 809, 817.
- „ phthisis, 584.
- „ rheumatic arthritis, 312.
- „ rheumatic fever, 299.
- „ rickets, 317.
- „ scurvy, 386.
- „ small-pox, 156.
- „ typhoid fever, 70, 71.

Digestive system, diseases, 608.

Digestion, 628.

- „ acids and alkalis, 629.
- „ albumoses and peptones, 629, 645.
- „ disturbances, 630, 631, 632.
- „ ferments, 628, 629, 630.
- „ gastric, 629.
- „ intestinal, 629.
- „ oral, 628.
- „ pancreatic, 629.

Diphtheria, 92.

- „ albuminuria, 788.
- „ animals, lower, 94.
- „ anterior horn cells, 914.
- „ antitoxin, 101.
- „ anuria, 780.
- „ bacillus, 93.
- „ bacteriological examination, 98.
- „ broncho-pneumonia, 96.
- „ cardiac failure, 97.
- „ cough, 102.
- „ deafness, 98.
- „ dyspnoea, 102.
- „ eye affections, 98.
- „ electrical reactions, 98.
- „ false membrane, 95, 96, 97, 102.
- „ fever, 96.
- „ hæmorrhage, 97.
- „ immunity, 94.
- „ laryngeal (abductor) paralysis, 1060.
- „ latent, 96.
- „ loss of smell and taste, 98.
- „ lymphatic glands, 95, 97.
- „ mania, 893.
- „ methods of spread, 93.
- „ milk as carrier, 93.
- „ myocardial degenerations, 96.
- „ nephritis, 96, 98, 798, 799, 802.
- „ neuritis, multiple, 98, 944, 1015.
- „ œdema glottidis, 497.

Diphtheria—

- „ outfits for swabs, 99.
- „ paralysis, 98.
- „ pulmonary collapse, 96.
- „ pulmonary interstitial emphysema, 96, 519.
- „ relapse, 97.
- „ spleen, 29.
- „ stridor, 102.
- „ staphylococcus and streptococcus, 96.
- „ swabs of throat, 99.
- „ syncope, 96.
- „ tendon reflexes, 98.
- „ third nerve neuritis, 1037.
- „ toxin, 95, 97, 100.
- „ urine, 98.
- „ voice, 97.

Diphtheritic enteritis, 683.

- „ gastritis, 649.
- „ membrane in throat, 95, 96, 102.
- „ paralysis, 98.
- „ treatment, 101, 1042.

Diphtheroid bacillus in general paralysis of insane, 946.

Diplegia, infantile, 924.

Diplococcus intracellularis meningitidis of Weichselbaum and of Still, 77, 853.

Diplococcus lanceolatus of Fränkel (of pneumonia), 535.

- „ meningitis, 853.
- „ pleurisy, 599.
- „ peritonitis, 759.

Diplococcus rheumaticus, 294.

"Dippers" disease, 288.

Dissecting aneurism, 481.

Disseminated sclerosis, 948.

- „ intention tremor, 950.
- „ nystagmus, 950.
- „ speech, 950.

Distoma Ringeri, 254.

Distomiasis, 254.

- „ ascites, 254.
- „ bathing, 255.
- „ cystitis, 255.
- „ diarrhoea, 255.
- „ hæmaturia, 255.

Dittrich's plugs, 513.

Diver's paralysis, 882.

Diverticulum of intestine (Meckel's), 672.

- „ œsophagus, 624.

Dochmius duodenale, 264.

Double vision, in fourth nerve paralysis, 1039.

Double Vision—

- „ sixth nerve paralysis, 1040.
- „ tabes dorsalis, 935.

Dracontiasis, 268.

Dracunculus medinensis, 268.

Dreamy state, as an aura in epilepsy, 972.

Drowsiness after epileptic fit, 972.

- „ phosphorus poisoning, 287.

Dropsy, 4.

- „ anæmia (pernicious), 356.
- „ beri-beri, 212.
- „ cardiac, 428, 447, 448, 460.
- „ cerebral ventricles (hydrocephalus), 5, 858.
- „ chlorosis, 350.
- „ filariasis, 270.
- „ gastric cancer, 665.
- „ hepatic cancer, 729.
- „ hepatic cirrhosis, 715, 718, 721.
- „ lungs, 527.
- „ nephritis, acute, 803.
- „ „ chronic interstitial, 815.
- „ „ chronic parenchymatous, 808, 809.
- „ nomenclature, 4, 5.
- „ obstructive, 6.
- „ pericardium (hydropericardium), 4, 410.
- „ peritoneum (ascites), 4, 755.
- „ pleura (hydrothorax), 4, 594.
- „ scarlet fever, 168.
- „ subcutaneous tissues (anasarca), 5.
- „ tunica vaginalis testis (hydrocele), 5.

Dropsical fluid, causes and composition, 5.

Drunken state, diagnosis from apoplexy, 876.

Dry cough in adenoids, 495.

- „ „ pleurisy, 602.

Dry mouth and throat in adenoids, 495.

Ductless glands, 389.

Ductus arteriosus, patent, 409.

Dulness, wooden in pleurisy, 603.

- „ pneumonia, 539.

Dumb rabies, 228.

Duodenal ulcer, 660.

Duodenitis, 679.

Duodenum, cancer, 698.

- „ inflammation, 679, 738.
- „ ulcer, 660.

- Dura mater, endothelioma, 848.
 " " hæmorrhage, 844.
 " " inflammations, 844.
 " " tumors, 848.
 Dust inhalation diseases, 555.
 " particles and tubercle bacillus, 110.
 Dysarthria, 961.
 Dysentery, 207.
 " amœba, 208, 209, 711.
 " bacillus coli, 208.
 " bacillus, specific (Shiga's), 208.
 " catarrhal, 207.
 " colitis, ulcerative, 684.
 " diphtheritic, 207.
 " hepatic abscess, 209, 710.
 " ipecacuanha, 210.
 " stools, 209.
 " stricture, intestinal, 674.
 " tenesmus, 209.
 " torminia, 209.
 " tropical, 209.
 " ulceration, 208.
 Dyspepsia, 628.
 " acidity, 631, 632.
 " borborygmi, 632.
 " gastric, 631, 632.
 " intestinal, 632.
 " pancreatic, 632.
 " pyrosis, 632.
 " table, 634.
 " waterbrash, 632.
 Dysphagia, in aneurism of aorta, 482.
 " hydrophobia, 229.
 " hysteria, 988.
 " laryngeal tumors, 502.
 " œsophageal cancer, 627.
 " diverticulum, 624.
 " stenosis, 625.
 " scarlet fever, 167.
 Dyspnœa, in aneurism (thoracic), 482.
 " anæmia, pernicious, 356.
 " aortic incompetence, 453.
 " cardiac dilatation, 428.
 " chlorosis, 351.
 " diabetes mellitus, 334.
 " emphysema, 520.
 " exophthalmic goitre, 399, 407.
 " fatty heart, 418.
 " gout, 322.
 " laryngitis, 102, 497.
 " laryngismus stridulus, 502.
 " lymphadenoma, 378.

- Dyspnœa—
 " mitral incompetence, 447, 448.
 " œdema glottidis, 498.
 " pericardial effusion, 414.
 " phthisis, 578, 579.
 " tuberculosis, acute miliary, 553.
 " uræmia, 783.
 " whooping-cough, 178.
 Dystrophies, muscular, 921.
 Ear disease, in abscess of brain, 893.
 " " scarlet fever, 168.
 Earthy complexion in chronic Bright's disease, 828.
 Eberth's bacillus, 59.
 Echinococcal cyst, 259.
 Echinococcal disease, 258.
 Echolalia, 970.
 Eclampsia, 977.
 Ectocyst of a hydatid cyst, 259.
 Ectopia cordis, 408.
 Ectropion in facial paralysis, 1051.
 Eczema of tongue, 609.
 Efferent system, 834.
 Eighth nerve, 1054.
 " cochlear branch, 1054.
 " tinnitus aurium from lesion, 1055.
 " vestibular branch, 1056.
 Elastic fibres in sputum, 577.
 Electrolysis in aneurism, 486.
 " hydatid cysts, 730.
 Elephantiasis, arabum, 271.
 " Græcorum, 123.
 Elephantoid fever, 271.
 Emaciation, in anorexia nervosa of hysteria, 988.
 " gastric cancer, 664.
 " œsophageal cancer, 627.
 " phthisis, 578.
 Embolic abscess, 137.
 " theory of gastric ulcer, 653.
 Embolism, 10.
 " brain, 884.
 " coronary arteries, 424.
 " endocarditis (malignant), 439, 441.
 " intestine, 679.
 " kidney, 778.
 " spleen, 12.
 Emboli, multiple, in lung, 11, 531.
 Embryonic residue theory of Cohnheim, 39.
 Emphysema, pulmonary, 518.
 " apex of heart, 521.
 " asthma, 517.

Emphysema—

- „ barrel chest, 520.
 - „ bronchiectasis, 512.
 - „ bronchitis, 507, 509, 520.
 - „ collapse, pulmonary, 524.
 - „ compensatory, 520.
 - „ cough, 520.
 - „ cyanosis, 520.
 - „ diphtheria, 96, 519.
 - „ dropsy, 520.
 - „ dyspnoea, 520.
 - „ heart, hypertrophy and dilatation of right side, 426, 520.
 - „ hyperresonance, pulmonary, 521.
 - „ interstitial, 519.
 - „ local, 519.
 - „ phthisis, chronic, 575.
 - „ pneumonia, lobular, 545.
 - „ pulmonary second sound, accentuated, 521.
 - „ senile, 520.
 - „ substantial, 520.
 - „ miliary tuberculosis of children, 561.
 - „ vesicular, 519.
 - „ whooping-cough, 179.
- Empyema of antrum, 1005.
- „ gall bladder, 732.
 - „ pleural cavity, 601.
- Empyema (thoracic), 601.
- „ albumosuria, 789.
 - „ bacteria, 601.
 - „ burrowing, 601.
 - „ encysted form, 549.
 - „ excision of ribs, 605.
 - „ fever, 603.
 - „ gall bladder, 732.
- Encephalitis, acute, 891.
- „ abscess, 892, 893.
 - „ chronic, 896.
 - „ diffuse, 892.
 - „ focal, 892.
 - „ meningitis, 851, 892.
 - „ sclerosis, 892.
 - „ softening, 892.
 - „ suppurative, 893.
- Endarteritis obliterans, 476.
- Endocarditis, 431.
- „ acute, 432.
 - „ bacteria, 432, 433, 434, 436, 439.
 - „ benign, 435.
 - „ chorea, 434.
 - „ chronic, 442.
 - „ „ alcoholism, 443.

Endocarditis—

- „ calcification, 443.
 - „ embolism, 437, 439.
 - „ foetal, 409.
 - „ gonorrhœa, 435.
 - „ gout, 443.
 - „ incompetence, 443.
 - „ infective, 438.
 - „ influenza, 435.
 - „ left side, 438.
 - „ malignant, 438.
 - „ peliosis rheumatica, 435.
 - „ pneumonia, 435, 540.
 - „ primary, 433.
 - „ purpura, 435.
 - „ rheumatism, acute, 296, 434.
 - „ right side, 438.
 - „ secondary, 433.
 - „ simple, 435.
 - „ stenosis, 443.
 - „ strain, 443.
 - „ syphilis, 443.
 - „ suppurative, 438.
 - „ tonsillitis, 435.
 - „ tuberculosis, 435.
 - „ vegetations, 434, 435, 436, 438.
 - „ warty, 435.
- Endocarditis, malignant, 438.
- „ abscess of lung, 549.
 - „ apoplexy, 439.
 - „ arthritis, 440.
 - „ cardiac, cases of, 439.
 - „ cryptogenetic, 439.
 - „ embolism of brain, kidney or spleen, 439.
 - „ gonorrhœa, 305, 435.
 - „ meningitis, 853.
 - „ myocarditis, 439.
 - „ pyæmia, 440.
 - „ typhoid fever, 440, 441.
- Endocyst of a hydatid cyst, 259.
- Endogenous hydatid cysts, 259.
- Endothelioma of lung, 592.
- „ salivary glands, 616.
- Enteralgia, 704.
- Enteric fever, 58 (*see* typhoid fever).
- Enteritis, anæmia and, 682.
- „ animal parasites and, 682.
 - „ bacteria, 681, 682.
 - „ catarrhal, 683.
 - „ children, 681, 685.
 - „ colitis, mucous, 684.
 - „ „ ulcerative, 684.
 - „ convulsions, 685.
 - „ croupous, 683.
 - „ diarrhœa, 684, 685.

Enteritis—

- „ diphtheritic, 683.
 - „ fever, 685.
 - „ fibrinous, 683.
 - „ follicular, 683.
 - „ fruit, unripe, 681, 685.
 - „ hepatic cirrhosis, 683.
 - „ membranous, 684.
 - „ mucus, 684, 685.
 - „ nephritis, 682, 683.
 - „ pains, 685.
 - „ phlegmonous, 683.
 - „ seasonal variation, 687.
 - „ ulcerative, 683.
 - „ vomiting, 685.
 - „ waxy degeneration, 775.
- Entero-colitis of cholera infantum, 681.

Enteroptosis, 637, 772.

Enumeration of blood-cells, 341.

Eosinophilia, ankylostomiasis, 347, 265.

- „ asthma, 347, 515.
- „ gout, 347.
- „ leukæmia, 363.
- „ pemphigus, 347.
- „ syphilis, 347.
- „ trichinosis, 267, 347.

Ependyma of ventricles, granularity of in general paralysis, 945.

„ maceration of in tubercular meningitis, 851.

Ephemeral fever, 144.

Epidemic rose rash, 175 (*see* German measles).

Epididymitis, tubercular, 818.

Epilepsy, 970.

- „ age, 970.
- „ alcoholism, 971.
- „ aura, 971.
- „ bromides, 976.
- „ consciousness, 972.
- „ heredity, 971.
- „ Jacksonian, 838, 971, 974.
- „ major, 971.
- „ minor, 973.
- „ nocturnal, 974.
- „ reflexes, 972.
- „ stages, 971.
- „ status epilepticus, 973.
- „ syphilis, 971.
- „ trismus, 1047.

Epileptic fits, 972.

- „ hemiplegias of children, 888.
- „ sleeping sickness, 253.

Epistaxis, 493.

- „ adolescents, healthy, 494.
 - „ ascents, high, 494.
 - „ diphtheria, 97.
 - „ hæmophilia, 380.
 - „ granular kidney, 813, 815.
 - „ leukæmia, 368.
 - „ liver cirrhosis, 494, 720.
 - „ night blindness, 385.
 - „ predilection, site of, 494.
 - „ scarlet fever, 165.
 - „ scurvy, 385.
 - „ typhoid fever, 61, 493.
 - „ valvular heart disease, 494.
 - „ whooping-cough, 179.
- Erb's varieties of dystrophies (muscular), 921.
- „ paralysis, 1023.
 - „ syphilitic spinal paralysis, 927.

Ergotism, 279, 281.

Eructations, acrid, in dyspepsia, 631.

Eruptive fevers, 146.

Erysipelas, 139.

- „ albuminuria, 141.
- „ cellulitis, 140.
- „ chronic variety, 141.
- „ Coley's fluid, influence on malignant growths, 141.
- „ delirium, 141.
- „ diarrhoea, 141.
- „ facial, 141.
- „ stomatitis, suppurative, 613.
- „ fever, 141.
- „ infants, 142.
- „ insomnia, 143.
- „ iodine, 142.
- „ lymphatics, 140.
- „ meningitis, 141, 853.
- „ micro-organism, 140.
- „ œdema glottidis, 497.
- „ phlegmonous form, 143.
- „ phlegmonous gastritis, 649.
- „ streptococcus, 140.
- „ thrombosis of cerebral sinuses, 889.
- „ urine, 141.
- „ wound and, 141.

Erythema nodosum in purpura, 383.

Erythrocytes, 342.

Erythromelalgia, 999.

État mammeloné of stomach, 642.

État ségmentaire in myocardium, 420.

European cholera, 84.

Exaltation, mental, in general paralysis of insane, 947.

- Exanthemata, 146.
 Exogenous hydatid cysts, 259.
 Exophthalmic goitre, 396.
 " asthenia, 399.
 " cornea, 398.
 " dyspnœa, 399, 407.
 " hair, 399.
 " insomnia, 399.
 " lymphatic glands, 397.
 " mania, 399, 893.
 " palpitation, 398.
 " pigmentation, 399.
 " proptosis, 398.
 " Stellwag's sign, 398.
 " tachycardia, 398.
 " tremors, 398.
 " thymus gland, 398.
 Exophthalmos in exophthalmic goitre, 398.
 " congenital hydrocephalus, 850.
 Eye muscles, paralysis, 1037, 1041.
 " spasm, 1042.
 Eyes, conjugate deviation, 1042.
 " apoplexy, 872.
 Facial hemianæsthesia in paralysis of sensory part of fifth nerve, 1045.
 " hemiatrophy, 1000.
 " leonine, in leprosy, 125.
 " mask-like in paralysis agitans, 963.
 " nerve, 1048.
 Facies, choleric, 83.
 " hepatic, 721.
 " hippocratic, 760.
 " myxœdema, 394.
 " pinched in peritonitis, 763.
 " syphilis, hereditary, 189.
 " vacuous in adenoids, 495.
 Fæcal accumulations, 682.
 " concretions, 687.
 " vomiting (stercoraceous), 675.
 Fallopian canal, course of seventh nerve, 1049.
 Fallopian tubes, inflammation, 758, 767.
 " tuberculosis, 818.
 Famine fever, 52 (*see* relapsing fever).
 Farcy, 219.
 " buds, 220.
 Fat embolism in diabetes mellitus, 329.
 Fat necrosis in acute pancreatitis, 21, 750.
 Fat in stools, 746.
 " urine, 747.
 Fatty degeneration, 24.
 " arteries, 474.
 " heart, 417.
 " kidney, 773.
 " liver, 707.
 " pancreas, 747.
 Fatty diarrhœa, 681, 747.
 " infiltration, 31.
 " heart, 418.
 " liver, 707.
 Febricula, 144.
 " children, 144.
 " delirium, 145.
 " epistaxis, 145.
 " herpes, of mouth, 145.
 Febrifuges, 50.
 Fehling's test for sugar, 330.
 Fermentation test for sugar, 331.
 Ferrier's snuff, 490.
 Festinant gait of paralysis agitans, 964.
 Fetid bronchitis, 508.
 " sputum in gangrene of lung, 553.
 " stomatitis, 611.
 Fever, 41, 43.
 " bilious remittent, 244.
 " blackwater, 246.
 " breakbone, 205.
 " British yellow, 204.
 " camp, 192.
 " causation, 45.
 " cerebro-spinal, 77.
 " clinical types, 46.
 " continuous, 46.
 " critical evacuation, 44.
 " dandy, 205.
 " Danubian, 87.
 " degenerative changes, 44.
 " diet, 50, 51.
 " elephantoid, 271.
 " enteric, 58.
 " ephemeral, 144.
 " eruptive, and their rashes, 147.
 " famine, 52.
 " furnishing of sick room, 49.
 " gastric, 639.
 " hæmoglobinuric, 246.
 " hysterical, 989.
 " infantile remittent, 73.
 " intermittent, 46, 88, 242.
 " lung, 535.
 " Malta, 87.
 " malignant purpuric, 77.

Fever—

- „ medicines, 60.
- „ Mediterranean, 87.
- „ Neapolitan, 87.
- „ nutrition, 44.
- „ pernicious, 242.
- „ production, 45.
- „ puerperal, 143.
- „ relapsing, 46, 52.
- „ remittent, 46, 242.
- „ rheumatic, 293.
- „ rock, 87.
- „ respiratory changes, 44.
- „ scarlet, 163.
- „ seven day, 52.
- „ ship, 192.
- „ simple continued specific, 46.
- „ splenic, 222.
- „ spirilla, 52.
- „ stages, 43, 44.
- „ temperature, 43.
- „ types, clinical, 46.
- „ treatment, general, 47-51.
- „ tuberculosis, course of, 119, 578.
- „ typhoid, 58.
- „ typhus, 201.
- „ undulant, 87.
- „ urine, changes, 44.
- „ zymotic, 146.

Fibrin ferment, source of, 18.

Fibrinous bronchitis, 509.

„ enteritis, 683.

„ gastritis, 649.

„ pharyngitis in diphtheria, 97.

„ pneumonia, 534.

Fibroid, heart, 419.

„ phthisis, 575.

Fibroma, 38.

„ lung, 591.

Fibrosis, arterio-capillary, 477.

„ brain, 892.

„ liver, 713.

Fidgets, 966.

Fifth nerve, 1043.

„ affections of sensory portions, 1045.

„ ageusia, 1048.

„ Gasserian ganglion, 1044.

„ pontine lesions, effect of, 1046.

„ spasm of masticatory muscles and, 1047.

Filaria Bancrofti, 270.

„ diurna, 271.

„ loa, 271.

Filaria—

„ nocturna, 269.

„ perstans, 271.

Filariasis, 269.

„ chylous ascites, 270.

„ chyluria, 270, 271.

„ lymph scrotum and varix, 270.

Fingers, clubbing of, in pulmonary osteo-arthritis, 1002.

Fish, putrid, and leprosy, 124.

Fistula in ano, 695.

„ gastro-coli in gastric cancer, 664.

„ „ ulcer, 656.

„ rectal, 680, 695.

Flat foot and metatarsal neuralgia, 1008.

Flatulence in dyspepsia, 632.

Flies and typhoid fever, 59.

Flint's murmur in aortic incompetence, 453.

Floating kidney, 771.

Flukes, 254 (*see* distomiasis).

Fetal endocarditis, 409, 432, 457.

Follicular tonsillitis, 619.

Food poisoning, 279.

„ cholera-like symptoms, 85.

„ coma, 279.

„ convulsions, 279.

„ prostration, 279.

Foot and mouth disease, 234.

Foot-drop from paralysis of external popliteal branch of sciatic nerve, 1018, 1027.

Foramen ovale, patency of, 408, 457.

Foreign bodies in appendix, 687.

„ in bronchus, 517.

Fourth nerve, lesions, 1039.

Fremitus, friction in pleurisy, 603.

„ hydatid, 260.

„ vocal, 539, 579, 603.

Friction, pericardial, 414.

„ pleural, 603.

Friedreich's ataxia, 941.

„ age, 941.

„ gait, 942.

„ nystagmus, 942.

„ Romberg's sign, 942.

„ sclerosis, 942.

Fulminating appendicitis, 692.

„ meningitis, 855.

„ purpura, 383.

Functional albuminuria, 787.

„ brain diseases, 962.

„ cardiac disorders, 461.

Functional albuminuria—

- „ cardiac incompetence, 446.
- „ liver disturbance, 810.

Fungus ray, 129 (*see actinomyces*).

Funnel-shaped mitral, 443.

Gait, ataxic, in ataxic paraplegia, 940.

- „ „ locomotor ataxia, 934.
- „ drunken, in alcoholism, 275.
- „ cerebellar tumor, 943, 956.
- „ Friedreich's ataxia, 940.
- „ festinant, in paralysis agitans, 964.
- „ groggy, in cerebellar ataxia, 943.
- „ high-steppage, in multiple neuritis, 1016.
- „ „ paralysis of external popliteal nerve, 1027.
- „ pseudo-hypertrophic paralysis, 922, 923.
- „ primary and secondary spastic paralysis of adults, 926.
- „ slow and slouching in acromegaly, 1000.

Gall bladder, aspiration of, 737.

- „ cancer, 743.
- „ dilation, 733.
- „ diseases, 730.
- „ empyema, 732.
- „ inflammation, 732, 737.
- „ peritonitis, 758.
- „ shrinking, 733.
- „ tumor (pear-shaped) of abdomen in dilatation, 733.

Gallop cardiac rhythm in granular kidney, 815.

Galloping consumption, 564.

Gall stones, 730.

- „ abscesses, 733.
- „ age, 731.
- „ bacteria, 730, 731.
- „ bile duct obstruction, 733.
- „ cancer, 732, 744.
- „ characters and composition, 731, 732.
- „ cholangitis, 733, 738.
- „ cholecystitis, 733, 737.
- „ colic fever, 734.
- „ fever, 734.
- „ intestinal obstruction, 734.
- „ jaundice, 733.
- „ moving, 732.
- „ pancreatitis, acute, 749.
- „ rigors, 734.

Gall stones—

- „ sex, 731.
- „ stationary, 732.

Ganglia, basal lesions, 838.

Ganglia, spinal, in tabes dorsalis, 929.

Gangrene, 21.

- „ cheek, 612.
- „ diabetes mellitus, 335.
- „ lung, 552.
- „ Raynaud's disease, 996, 998.
- „ symmetrical, 996.

Gangrenous stomatitis, 612.

Garrod's thread test for uric acid, 322.

Gasserian ganglion, extirpation of, in tic douloureux, 1010.

Gastralgia, 670.

Gastrectasis, 650, 652.

Gastric catarrh, simple acute, 638.

- „ children, aphasia, asthma and fits in, 639.

„ fever, 639.

„ diminution of hydrochloric acid, 639.

„ pain in, 639, 640.

„ ulcers, 639.

„ vomiting, 639.

Gastric catarrh, simple chronic, 641.

„ anæmia, 645.

„ appetite, 643.

„ chloride excretion, diminished, 645.

„ dilatation of stomach, 644, 651.

„ discomfort, 643.

„ erosions, hæmorrhagic, 642.

„ état mammeloné, 642.

„ flatulence, 644.

„ hæmatemesis, 645.

„ hydrochloric acid, diminished, 645.

„ lavage, 647.

„ leather bottle stomach, 643.

„ mucus, excess, 642, 645.

„ pain, 644.

„ sclerosis, 643.

„ tenderness, epigastric, 642.

„ ulcer, gastric, 642, 655.

„ vertigo, 645.

„ vomiting, 644.

Gastric catarrh, simple subacute, 640.

„ appetite, 641.

„ flatulence, 641.

„ headache, 641.

Gastric catarrh—

- „ hydrochloric acid, diminished, 641.
- „ pain, 641.
- „ stools, clay-coloured, 641.
- Gastric crises in tabes dorsalis, 935.
- „ fever, 639.
- Gastric juice, abnormal acids, presence, 631, 645, 665.
- „ ferments, 629.
- „ hydrochloric acid, diminution of in cancer, 631, 665.
- „ acute, 639.
- „ subacute, 641.
- „ later chronic gastric catarrh, 645.
- „ excess of in early chronic gastric catarrh, 644.
- „ tabes dorsalis in gastric ulcer, 631, 658.
- „ mucus in chronic gastric catarrh, 645.
- „ pepsin, 629, 630.
- Gastric ulcer, 653.
- „ abscess in, metastatic, 657.
- „ „ subphrenic, 656, 657, 765.
- „ age, 655.
- „ anemia, 654, 657.
- „ chlorosis, 654.
- „ death, rapid, 656.
- „ fistulae, gastric, 656.
- „ hematemesis, 656, 657, 659.
- „ hour-glass stomach, 656.
- „ hydrochloric acid, excess, 658.
- „ melæna, 658.
- „ number, 655.
- „ pain, 657, 658.
- „ pericarditis, 657.
- „ peritonitis, 656, 657.
- „ pleurisy, 657.
- „ sex, 655.
- „ site, 655.
- „ size, 655.
- „ shock, 656.
- „ symptoms, absent, 659.
- „ vomiting, 657.
- Gastric vertigo, 1057.
- Gastritis, croupous, 649.
- „ diphtheritic, 649.
- „ infective, 638, 649.
- „ membranous, 638, 649.
- „ bacteria, 650.
- „ mycotic, 638, 649.
- „ phlegmonous, 638, 649.

Gastritis—

- „ polyposa, 642.
- „ simple, acute, 638.
- „ subacute, 640.
- „ chronic, 641.
- „ toxic, 648.
- Gastro-colic fistula, in gastric cancer, 664.
- „ „ gastric ulcer, 656.
- Gastrodynia, 670.
- Gastro-jejunoscopy in gastric ulcer, 659.
- Gastroptosis, 637.
- General paralysis of the insane, 944.
- „ age, 945.
- „ aphasia, 947.
- „ bedsores, 947.
- „ bacillus diphtheroides, 946.
- „ delirium, expansive, 947.
- „ eye symptoms, 947.
- „ melancholia, 947.
- „ pachymeningitis hæmorrhagica interna, 846, 945.
- „ sex, 945.
- „ speech, 946.
- „ syphilis, 945.
- „ tabes dorsalis, 945.
- „ tremor, 946.
- Genito-urinary tuberculosis, 818.
- Geographical tongue, 609.
- German measles, 175.
- „ desquamation, 177.
- „ fever, 176.
- „ incubation period, 175.
- „ itching, 176.
- „ rash, 176.
- „ sore throat, 176.
- Gigantism, 1001.
- Gilles de la Tourette's disease, 970.
- Gin-drinker's liver, 715.
- Glands, 219.
- „ abscesses, 221.
- „ agglutination test, 221.
- „ animals, 219.
- „ bacillus, 219.
- „ bacteriological diagnosis, 221.
- „ cell changes, 220.
- „ farcy buds, 220.
- „ infection, methods, 219.
- „ lymphadenitis and lymphangitis, 220.
- „ man, 219.
- „ mallein, 221.
- „ nasal discharge, 220.
- „ pneumonia, 220.
- „ suppuration, 220, 221.
- „ ulcers, 220.

- Glioma, 38.
 " brain, 952.
 Gliosis and syringomyelia, 860.
 Globulin in urine, 789.
 Globus hystericus, 986.
 Glomerular nephritis, 799, 802.
 Glossina palpalis and morsitans, 251.
 Glosso-labio laryngeal paralysis, 917.
 Glottidis œdema, 497.
 " " angio-neurotic œdema, 996.
 " " Bright's disease, 498, 803.
 " " diphtheria, 497.
 " " erysipelas, 497.
 " " heart disease, 498.
 " " laryngitis, syphilitic, 497, 500.
 " " tubercular, 497, 500.
 Glycogen, formation and transformation, 328.
 Glycogenesis, 328.
 Glycosuria, abnormal, 328.
 " alimentary, 334.
 " cerebellar tumor, 956.
 " diabetic, 330.
 " fourth ventricle, puncture, 327.
 " gout, 322, 335.
 " non-diabetic, 334.
 " Raynaud's disease, 998.
 " temporary, 334.
 " whooping-cough, 179.
 Glycuronic acid, 792.
 Gmelin's test for bile, 793.
 Goitre, 392.
 " cretinism, 395.
 " colloid, 392.
 " exophthalmic, 396.
 " parenchymatous, 392.
 Gonorrhœal arthritis, 305, 306.
 " conjunctivitis, 305.
 " endocarditis, 305, 435.
 " lymphadenitis, 305.
 " meningitis, 305.
 " rheumatism, 298, 304.
 Gout, 318.
 " arterial sclerosis, 322.
 " arthritis, 320, 321.
 " bronchitis, chronic, 507.
 " cartilage, 320.
 " chronic deforming, 320.
 " diet, 323, 324.
 " dislocation, 320.
 " dyspnœa, 322.
 " fever, 321.
 " glycosuria, 322, 335.
 " heredity, 318.
 " irregular, 321, 322.
 " kidney, 319, 322, 798, 811, 812.
 " leucocytosis, 319.
 " metastatic, 321.
 " metatarsophalangeal joint, 319.
 " pharyngitis, 617.
 " pre-gouty state, 784.
 " recurrence, 321.
 " regular, 321.
 " retrocedent, 321, 322.
 " sex, 318, 326.
 " rheumatic, 307.
 " rich living, 318.
 " thread test, 322.
 " tophi, 321.
 " typical attack, 321.
 " uric acid, 319, 784.
 Gouty kidney, 811, 812.
 Græfe's sign, 398.
 Grandeur, ideas of, in general paralysis of insane, 947.
 Grand mal, 971.
 Granular kidney, 809.
 Gravel, 797.
 Graves's disease, 396.
 Greenish hue of face in chlorosis, 351.
 Gripes, 704.
 Grippe, 89.
 Guinea worm, 268.
 " abscess, 269.
 " crustaceans, 268.
 Gumboil, 613.
 Gumma, 185, 187.
 " brain, 952.
 " heart, 423.
 " liver, 719.
 " lung, 590.
 " miliary in hereditary syphilis, 719.
 " pharynx, 614.
 Gunmatous formation in cerebral arteries, 188.
 Gummy tumors, 187.
 Gums, absorption of, in pyorrhœa alveolaris, 611.
 " bluish-black line in lead poisoning, 290.
 " red and swollen in fetid stomatitis, 611.
 " spongy in scurvy, 385.
 Gustatory sense, abolition, 1048.

Habit spasm, 970.

Hæmatemesis, 12, 671.

,, enlarged spleen, 671.

,, gastric cancer, 665.

,, " catarrh, 645.

,, " congestion, 671.

,, " ulcer, 656, 657, 659.

,, hæmoptysis, comparison with, 13.

,, hepatic cirrhosis, 720.

,, " congestion, 708.

,, " yellow atrophy, 726.

,, purpura, 383, 671.

,, scurvy, 385, 671.

,, treatment, 14.

Hæmatinuria, 388.

Hæmatogenous jaundice, 740.

Hæmatoidin, 31, 870.

Hæmatoma, of brain, 870.

,, of cord, 881.

,, of dura, 846.

Hæmatomyelia, 881.

,, Brown-Séquard's paralysis, 882.

Hæmatoporphyrin in urine, 786.

Hæmatorrhachis, 879.

,, death from, 880.

,, herpes, 880.

,, paralysis, 880.

Hæmatozoon of hæmoglobinuric fever in cattle, 247.

Hæmaturia, 790.

,, bilharzia, 255, 790.

,, bladder (urinary) disease, 791.

,, carbolic acid, 790.

,, filaria sanguinis hominis, 790.

,, hydronephrosis, 822.

,, injuries, 790.

,, nephritis, acute, 803.

,, " chronic interstitial, 815.

,, " chronic parenchymatous, 80.

,, renal calculus, 790, 821.

,, " cancer, 827.

,, " congestion, 777.

,, " infarction, 778.

,, " tubercle, 819, 821.

Hæmochromatosis, 752.

Hæmocytometer, 341.

Hæmodynamometer, 473.

Hæmoglobin, estimation, 341.

,, reduction of, in chlorosis, 351.

Hæmoglobinometer, 341.

Hæmoglobinuria, 791.

,, from antifebrin, 388.

,, burns, 791.

,, cold, 791.

,, drugs, 791.

,, horses, 388.

,, paroxysmal, 388, 791.

,, Raynaud's disease, 388, 792, 998.

,, septicæmia, 791.

,, toxic, 248, 791.

Hæmoglobinuric fever, 246.

,, anæmia, 247.

,, anuria, 248.

,, fever, 247.

,, hæmatozoon, 246, 247.

,, jaundice, 247.

,, leucocytosis, 247.

,, pigment, 247.

,, quinine, 246.

,, renal casts, 247.

,, rigors, 247.

,, urine, 247.

,, uræmia, 248.

Hæmolysis, from antifebrin, 388.

,, burns, 387.

,, cholangitis, 740.

,, horses, 388.

,, toxic, 388.

Hæmopericardium, 410.

Hæmophilia, 379.

,, epistaxis, 380.

,, heredity, 379.

,, joints, 380.

,, sex, 379.

Hæmoptysis, 528.

,, aneurismus, thoracic, 483, 529.

,, apoplexy of lung, 531.

,, bronchiectasis, 513.

,, causes, 528.

,, diphtheria, 97.

,, distoma Ringeri, 254.

,, fibrinous bronchitis, 510.

,, gangrene, pulmonary, 553.

,, health, 529.

,, heart disease, 446, 529, 531.

,, hydatids of lung, 593.

,, lung, infarcts, 530.

,, mountain ascents, 529.

,, phthisis, chronic, 577, 588.

,, purpura hæmorrhagica, 383, 528.

,, treatment, 14, 531.

,, tuberculosis of lung, 529, 531, 569, 573.

- Hæmorrhage, 12.
 „ adrenals, 402.
 „ anæmia, pernicious, 356.
 „ aneurism of aorta, 483
 „ „ cerebral arteries, 888.
 „ brain, 869.
 „ bronchiectasis, 513.
 „ cerebral, 867.
 „ diphtheria, 97.
 „ dura mater, 844
 „ encephalitis, 891, 892.
 „ hæmophilia, 380.
 „ hepatic cirrhosis, 720.
 „ „ yellow atrophy, 727
 „ Hodgkin's disease, 378.
 „ icterus neonatorum, 742
 „ leukæmia, 368.
 „ lung, 528.
 „ myxœdema, 394.
 „ nephritis, chronic, 807.
 „ nose, 493.
 „ optic neuritis, 1032.
 „ pancreas, 748.
 „ pigments, 34.
 „ plague, 216, 217.
 „ purpura, 383.
 „ relapsing fever, 54.
 „ scurvy, 385.
 „ „ infantile, 387
 „ spinal cord, 879
 „ tendency to, increased in jaundiced patients, 737.
 „ typhoid fever, 61, 62, 65, 67, 74.
 „ varicose veins, 4.
 „ ventricles of brain, 880.
 „ whooping-cough, 179
 „ yellow fever, 203.
 Hæmorrhagic ascites, 760, 770.
 „ bowel discharge in intus susception, 673.
 „ diseases, 379.
 „ glands in plague, 215.
 „ infarct, 12.
 „ lesions in anthrax, 224.
 „ small-pox, 149.
 „ yellow fever, 203
 Hæmorrhoids in hepatic cirrhosis, 718, 720.
 Hæmosiderin, 34, 35.
 Hæmothorax, 595.
 Haffkine's vaccine, in cholera, 83
 „ „ plague, 218.
 Hairy heart, 413.
 Hallucinations in delirium tremens, 276.
 Hanot's diabète bronze, 330.
 „ hypertrophic cirrhosis of liver, 715.
 Hay fever, 491
 „ bronchial asthma, 492.
 Headache from brain tumor, 952.
 „ cerebral abscess, 895.
 „ meningitis, 852.
 „ sick, 983.
 „ uræmia, 782.
 Head shaking, in paralysis agitans, 953.
 Heart, aneurism, 419, 424, 429
 „ apex of, in aortic incompetence, 452.
 „ „ hypertrophy, 427.
 „ „ mitral incompetence, 447.
 „ „ pericarditis, acute, 414.
 „ arrhythmia, 463.
 „ athlete's, 462.
 „ atrophy, 421.
 „ calcification in valves, 443.
 „ compensation in disease, 445
 „ dilatation of, 427.
 „ état ségmentaire, 420.
 „ exophthalmic goitre, 398.
 „ fatty, 418.
 „ „ anginoid pains, 418.
 „ „ death, sudden, 418, 419.
 „ delirium, 463.
 „ displacement of, in pleurisy, 603
 „ functional diseases, 461.
 „ hairy, 413
 „ hypertrophy, 425, 813.
 „ irritable, 452.
 „ malformations, 408
 „ malpositions, 408
 „ milk, spots on, 413
 „ Nauheim treatment, in diseases, 420
 „ neuroses, 461
 „ palpitation, 463.
 „ rapid, 464.
 „ rupture, 419, 421, 430
 „ slow, 466
 „ tobacco, 286, 463
 „ valvular diseases of, 432, 443
 „ weight, 426.
 Heat centres, 42.
 „ exhaustion, 292.
 „ loss, 42
 „ production, 42.

Heberden's nodes, 310.
 Hemeralopia, 1035.
 Hemianæsthesia, in cerebral hæmorrhage, 874.
 " hysteria, 987.
 " lesions of posterior third of internal capsule, 954.
 " paralysis of sensory part of fifth nerve, 1045.
 Hemianopia, in brain tumors, 954, 955.
 " chiasma lesions, 1036.
 " heteronymous, 1034.
 " homonymous, 1034.
 " migraine, 983.
 " nasal, 1036.
 Hemiataxia, 955, 956.
 Hemiatrophy, facial, 1000.
 Hemichorea, 966.
 Hemierania, 983.
 Hemipeptones, 629.
 Hemiplegia, 838, 873 (*see* cerebral hæmorrhage).
 " brain tumors, 954.
 " children, 886.
 " crossed, 874.
 " flasque, 875.
 " hysteria, 988.
 " œdema of brain, 867.
 " ordinary, 874.
 " Raynaud's disease, 998.
 Hemisection of spinal cord, 843.
 Hendon disease, 163.
 Henoch's purpura, 383.
 Hepatic abscess, alcoholic excess, 711.
 " amœba coli, 711.
 " bacteria, 711.
 " biliary, 711, 743.
 " colour of pus, 711.
 " cough, 712.
 " echinococcal, 711.
 " exploration of liver, 712.
 " fever, 711.
 " jaundice, 712.
 " leucocytosis, 712, 713.
 " multiple, 711, 713.
 " pyæmic, 713.
 " peritonitis, 758.
 " quinine, 712.
 " solitary, 711.
 " subphrenic abscess, 765.
 " tropical, 711.
 Hepatitis, acute, 709.
 " causes, 709.
 " chronic, 713-723.
 " interstitial, 713.

Hepatitis—
 " suppurative, 710.
 Hepatization of lung, 535.
 " grey and red, 535.
 Hereditary ataxia, 941.
 Heredity, in angio-neurotic œdema, 995.
 " cerebellar ataxia, 943.
 " colour blindness, 379.
 " diabetes insipidus, 338.
 " diabetes mellitus, 327.
 " disseminated sclerosis, 949.
 " epilepsy, 971.
 " Friedreich's ataxia, 941.
 " gout, 318.
 " hæmophilia, 379.
 " leukemia, 360.
 " nephritis, chronic, 812.
 " pseudo-hypertrophic paralysis, 921.
 " rheumatic fever, 293.
 " syphilis, 189.
 " Thomsen's disease, 1069.
 " tuberculosis, 110, 111.
 Herpes, in Bell's paralysis, 1052.
 " cerebro-spinal meningitis, 78.
 " frontalis, 1005.
 " hæmorrhachis, 880.
 " acute nasal catarrh, 489.
 " pneumonia, 538.
 " zoster, 938.
 Hiccough, 1021.
 " delirium tremens, 1022.
 " tabes dorsalis, 935.
 " treatment, 1022.
 " uræmia, 783.
 High tension pulse, 471.
 Hill-diarrhœa, 684.
 Hip-joint disease, and sciatica, 1007.
 Hippocratic facies, 750.
 Hodgkin's disease, 374.
 " anæmia, 377.
 " bone marrow, 377.
 " colour index, 377.
 " dyspnœa, 378.
 " fever, 378.
 " kidneys, 376.
 " leucocytosis, 377.
 " liver, 376.
 " lymphatic glands, 375.
 " lymphocytosis, 377.
 " spleen, 376.
 " thymus gland, 397.
 Homonymous image, 1041.
 "Horrors, the," 276.
 Hospital fever, 192.

- Hour-glass deformity of stomach, 637.
- "House to house visitation" in cholera, 85.
- Hunter's view of pernicious anæmia, 353.
- Huntingdon's chorea, 968.
- Hutchinson's teeth in syphilis, 189.
- " view of causation of leprosy, 124.
- Hyaline casts in urine, 794.
- Hydatids, 258.
- " brain, 952.
- " ecto- and endocyst, 259.
- " fluid, 259.
- " heart, 431.
- " hooklets, 259, 260.
- " liver, 260, 729.
- " lung, 593.
- " peritoneum, 729.
- " thrill, 260.
- Hydrocephalic cry, 852.
- Hydrocephalus, 5, 858.
- " external, 858.
- " " microcephalus, 858.
- " internal, 858.
- " " acute, 851, 858.
- " " angio-neurotic, 858.
- " " brain substance, 860.
- " " congenital, 858, 859.
- " " convulsions, 860, 861.
- " " exophthalmos, 860.
- " " face, 860.
- " " head, 860.
- " " imbecility, 860.
- " " lumbar puncture, 859-861.
- " " nystagmus, 860.
- " " optic neuritis, 861.
- " " sutures, cranial, 860.
- " " tumor (cerebral), 851.
- " " Wormian bones, 860.
- Hydrochloric acid of gastric juice, 631.
- " deficiency of, in gastric cancer, 668.
- " excess of, in gastric ulcer, 631.
- Hydromyelia, 862.
- Hydronephrosis, 822.
- " acquired, 822.
- " albuminuria, 823.
- " cardiac hypertrophy, 823.
- " congenital, 822.
- " hæmorrhage, 823.
- " intermittent, 823.
- " permanent, 822.
- Hydronephrosis—
- " polyuria, 823.
- " renal tuberculosis, 819.
- " uræmia, 823.
- Hydropericardium, 410.
- Hydrophobia, 225 (*see Rabies*).
- " pseudo, 230.
- Hydrops omenti, 756.
- Hydrothorax, 594.
- Hyperæmia, 4.
- Hyperæsthesia, in fifth nerve sensory division, lesions, 1046.
- " hæmatomyelia, 882.
- " hemisection or unilateral lesions of spinal cord, 844.
- " hysteria, 987.
- " myelitis, 901.
- " neurasthenia, 991.
- " rabies, 229.
- Hyperglycæmia in diabetes mellitus, 328, 329.
- Hypermetropia, a cause of spasm of ocular muscles and squint, 1042.
- Hypernephromata, 728, 827.
- Hypernephrotic cancer of liver, 728.
- Hyperosmia, 1029.
- Hyperplasia, 36.
- Hyperpyrexia, 43.
- " pneumonia, 43, 541.
- " rheumatic fever, 296, 300.
- " scarlet fever, 170.
- " sunstroke, 292.
- " treatment, 47, 48.
- " Teale's case, 43.
- " typhoid fever, 47, 48, 69, 73.
- Hyperresonance, in emphysema, 521.
- Hyperthermia, 46.
- Hypertrophic cirrhosis of liver, 715.
- Hypertrophy, 36.
- " compensatory, 37.
- " numerical, 36.
- " pseudo, 36.
- " pressure, 37.
- Hypoglossal nerve, 1066.
- Hypopion in small-pox, 154.
- Hypostatic congestion of lungs, 526.
- " pneumonias, 547, 549.
- Hypnotism in alcoholism, 278.
- Hysteria, 985.
- " abasia, 990.
- " adductor paralysis of larynx, 1061.
- " age, 985.

Hysteria—

- „ anæsthesia, 988.
- „ anorexia nervosa, 989.
- „ aphonia, 988, 989.
- „ arthritis, 988.
- „ astasia, 990.
- „ catalepsy, 987, 990.
- „ choreiform movements, 989.
- „ consciousness, 987.
- „ contractures, 988.
- „ fever, 990.
- „ globus hystericus, 986.
- „ heredity, 985.
- „ hyperæsthesia, 988.
- „ hypochondriasis, 990.
- „ hystero-epilepsy, 987.
- „ hystero-genetic areas, 987.
- „ insanity, 990.
- „ neurasthenia, 993.
- „ polyuria, 989.
- „ sex, 985.
- „ trance, 990.
- „ traumatic, 994.
- „ trismus, 1047.

Hysterical angina pectoris, or false
angina, 469.

Hysterical fever, 989.

Hystero-epilepsy, 987.

Hystero-genetic areas, 987.

Ice, typhoid bacilli, 59.

Icterus or jaundice, 34.

„ catarrhal, 738.

„ gravis, 724.

„ neonatorum, 34, 741, 745.

Icterus neonatorum, hæmorrhage
from umbilical cord, 741.

„ mild form, 742.

„ phlebitis, septic, 34, 709, 742.

„ syphilis, hereditary, 34, 741,
745.

Idiocy, birth palsies, 924.

Ileo-cæcal gurgling, 63.

Ileo-cæcal part of small intestine
most involved in typhoid
fever, 59.

Imbecility in birth palsies, 924.

Imitation chorea, 965.

Impotence in diabetes mellitus, 333.

„ dread of, in neurasthenia,
992.

„ tabes dorsalis, 935.

Impulsive tic, 971.

Inco-ordination in cerebellar tumors,
956.

„ chorea, 967.

„ tabes dorsalis, 933.

Indicanuria, 785.

Indigestion, 628 (*see* dyspepsia).

Indurative mediastino-pericarditis,
416.

Infantile convulsions, 977.

„ hemiplegia, 886.

„ paralysis, 904.

„ scurvy, 386.

„ spastic paralysis, 924.

Infarct, 12.

„ brain, 885.

„ heart, 424.

„ intestine, 678.

„ kidney, 778.

„ liver, 709.

„ lung, 530.

„ pancreas, 748.

„ spleen, 12.

Infective diseases, 52.

Inflammation, 14.

Inflation of bowel in intussuscep-
tion, 677.

Influenza, 89.

„ angio-cholitis, suppurative,
743.

„ arteritis, 476.

„ arthritis, 91.

„ bacillus, 89.

„ „ sputum, 90.

„ endocarditis, 433.

„ acute bronchitis, 503.

„ cardiac tonics, 92.

„ eye affections, 91.

„ gastro-intestinal form, 90.

„ mania, 90.

„ meningitis, 853.

„ nephritic form, 90.

„ nervous form, 90.

„ otitis media, 91.

„ pneumonia, 90, 546, 559.

„ pneumococcus, 90, 559.

„ pulmonary abscess, 560.

„ pulmonary gangrene, 560.

„ rheumatism acute, differen-
tiation, 91.

„ Russian, 89.

„ tuberculosis, 560.

Inhalation pneumonia, 555.

Inhibitory nerve of heart, 1061.

Inoculation, in cholera, 83.

„ hydrophobia, 231.

„ plague, 218.

„ small-pox, 160.

Insane, general paralysis of, 944.

Insect parasites, 273.

Insolation, 292.

„ fever, 292.

Insolation—

- „ respiratory paralysis, 292.
- Insomnia, in alcoholism, 275, 276, 278.
- „ cerebro-spinal meningitis, 78.
- „ diabetes mellitus, 333.
- „ exophthalmic goitre, 399.
- „ neurasthenia, 993, 994.
- „ pulmonary tuberculosis, 567.
- „ typhoid fever, 72.
- „ uræmia, 783.

Insular sclerosis, 948.

Intention tremor, 950.

Intercostal neuralgia, 1006.

Intermission of a cardiac beat, 463.

Intermittent fever, 45.

„ malarial, 242.

Internal capsule, lesions of, 954.

Interventricular cardiac septum, imperfections of, 409, 457.

Intestinal casts, 684.

„ colic, 704.

Intestinal obstruction, 672.

„ anæmia, 676.

„ anuria, 675.

„ constipation, 675.

„ diarrhœa, 676.

„ enemata, 677.

„ indicanuria, 785.

„ intussusception, 673.

„ pain, 675.

„ pseudo-diarrhœa, 675.

„ stricture, 674.

„ stools, 676.

„ tympanitis (abdominal), 675, 676.

„ volvulus, 674.

„ vomiting, bilious, 675.

„ „ gastric, 675.

„ „ stercoraceous, 675, 676.

Intestines, diseases of, actinomyces of, 130, 697.

„ amyloid degeneration, 678.

„ atony, 678, 682, 701.

„ atrophy, 678.

„ cancer, 675.

„ catarrh, 679, 683.

„ colic, 704.

„ congestion, 678, 683.

„ degenerations, amyloid, 576, 678, 775.

„ displacements, 672.

„ diverticulum of, 672.

„ hæmorrhage from, in typhoid fever, 62-74.

Intestines, diseases of—

„ hernia, 672.

„ hypertrophy, 678.

„ infarction, 678.

„ inflammation, 679.

„ intussusception, 672.

„ juice, digestive, 629.

„ malformation, 672.

„ obstruction, 672.

„ perforation of, in typhoid fever, 66, 67.

„ strangulation, 672, 673.

„ stricture, 674.

„ syphilis, 697.

„ tuberculosis, 694.

„ tumors, 674, 675.

„ ulceration, 682, 683.

„ volvulus, 674.

Intoxications, 26, 274.

Intra-labyrinthine pressure, interferences, 1056.

Intussusception, 672.

„ cause, 673.

„ gangrene, 674.

„ obstruction, 673.

„ peritonitis, 674.

„ stools, 673.

„ tumor, 674.

Intra-cranial tumors, 951 (*see* brain tumors).

Invaginated gut, 673.

Inverse type of temperature in tuberculosis, 119.

Iridocyclitis, in leprosy, 125.

Iridoplegia, 1039.

Iritis, in rheumatic fever, 295.

„ syphilis, 186, 189.

Iron pigment in pernicious anæmia, 355.

Irritable heart, 462.

Itch parasite, 272.

Itching of skin in jaundice, 739.

„ „ measles, German, 176.

Jacksonian epilepsy, 974.

Jail fever, 192.

Jaundice, angio-cholitis, 739, 741.

„ bile duct obstruction, 733, 734, 735, 745.

„ catarrhal, 738, 739.

„ cholæmia, 739, 741.

„ gall stones, 733.

„ hæmatogenous, 740.

„ hæmoglobinuric fever, 247.

„ hæmohepatogenous, 741.

„ hæmorrhage, tendency, 737, 739.

Jaundice—

- „ hemolysis, 743.
- „ infectious, 31.
- „ itching of skin, 739.
- „ liver abscess, 712.
- „ cancer, 729.
- „ cirrhosis, 715, 720, 721.
- „ yellow atrophy, 726.
- „ malaria, 741.
- „ neonatorum, 31, 709, 741, 745.
- „ obstructive, 31, 739, 740.
- „ pancreas, cancer, 753.
- „ pancreatitis, chronic, 752.
- „ phosphorus poisoning, 287, 741.
- „ pneumonia, 510.
- „ polycholia, 740.
- „ pruritus, 739, 741.
- „ relapsing fever, 53.
- „ suppression, 740.
- „ toxæmic, 740.
- „ toxic, 31.
- „ Weil's disease, 741.
- „ worms, 254, 262.
- „ yellow fever, 203, 741.
- „ yellow vision, 739, 741.

Jejunitis, 679.

Jenner's vaccination against small-pox, 159.

Kahler's disease, 789.

Kakke, 211.

Keratitis, leprosy, 125.

„ syphilis, 189.

Keratosis of feet and hands in arsenical neuritis, 1017.

Kernig's sign, 852.

Kidney, abscess, 801, 821.

„ atrophy, 823.

„ bimanual examination, 772.

„ calculi, 795.

„ cancers, 826.

„ cirrhosis, 807, 809.

„ congestion, 777.

„ cystic disease, 824.

„ degenerations, 773.

„ floating, 771.

„ gouty, 811, 812.

„ granular contracted, 809.

„ pale, 807.

„ horse-shoe, 771.

„ hypertrophy, 771.

„ hypernephromata, 827.

„ infarctions, 778.

„ inflammation, 798.

„ malformations, 771.

Kidney—

- „ malpositions, 771.
- „ movable, 771, 772.
- „ sarcoma, 826.
- „ sclerosis, 809.
- „ secretion, disturbances, 778.
- „ secretion, internal, 782.
- „ single, 771.
- „ syphilis, 773, 799, 818.
- „ tuberculosis, 818.
- „ tumors, 826.
- „ white (large), 774, 807.
- „ white (small), 807.

Kidney, movable, 771, 772.

„ enteroptosis, 772.

„ hæmaturia, 772.

„ hydronephrosis, 772, 823.

„ nephrorraphy, 773.

„ neurasthenia, 772.

„ sex, 772.

Kidney, surgical stripping of capsule in uræmia, 818.

Kidney, waxy, 773.

„ „ albuminuria, 775.

„ „ anæmia, 776.

„ „ apoplexy, 776.

„ „ casts, 775.

„ „ diarrhœa, 474, 776.

„ „ dropsy, 776.

„ „ polyuria, 474, 775.

Klebs-Löffler bacillus, 93.

Klumpke's paralysis, 1023.

Knee jerk, 840.

„ exaggerated in spastic paralysis, 926.

„ lost in tabes dorsalis, 933.

Koch's cholera bacillus, 80.

„ tuberculins, 122.

Kreatinin, 784.

Kyphosis in acromegaly, 1001.

Labyrinth, interferences of pressure within, 1056.

Lacunæ of Marie and cerebral hæmorrhage, 869.

Lacunæ of tonsils in follicular tonsillitis, 620.

Lænnec's atrophic cirrhosis of liver, 715.

Laminectomy in compression myelitis, 903.

Landry's paralysis, 910.

„ age, 910.

„ degenerations, 911.

„ fever, absent, 911.

„ hydrophobia, 229.

„ onset, 911.

- Landouzy-Dejerine, muscular dystrophy, 921.
 Langerhan's islands and diabetes, 747.
 Laparotomy, in hepatic cirrhosis, 723.
 „ typhoid perforation, 72.
 Laryngeal, crises, 935, 1061.
 „ diphtheria, 102.
 „ nerves, 1060.
 „ recurrent paralysis, 1060.
 Laryngismus stridulus, 502.
 „ child crowing, 502.
 „ cyanosis, 502.
 „ death, sudden, 502.
 „ muscular spasm, 1061.
 „ rickets, 316, 317, 502.
 Laryngitis, aphonia, 496, 500.
 „ adductor paralysis of larynx, 1061.
 „ catarrhal, acute, 102, 496.
 „ chronic, 498.
 „ cough, 102, 496, 500.
 „ diphtheria, 102.
 „ dyspnoea, 102, 497.
 „ dysphagia, 500.
 „ hypertrophic, 499.
 „ membranous, 102.
 „ oedematous, 497.
 „ sicca, 499.
 „ spasmodic, 102, 502.
 „ stenosis, 500.
 „ stridor, 103.
 „ syphilitic, 500.
 „ tubercular, 500.
 „ ulcerated, 500, 501.
 Larynx, diseases of, 496.
 „ abductor paralysis, 1061.
 „ adductor paralysis, 1061.
 „ muscular paralysis, 1060.
 „ spasm, 1061.
 „ paralysis (abductor) in diphtheria, 1060.
 „ paralysis (adductor) in hysteria, 1060.
 „ tumors, 501.
 „ ulcer of, in typhoid fever, 66.
 „ unilateral abductor paralysis, 1060.
 „ "Latent phase" of malaria, 244.
 Lateral sclerosis, amyotrophic, 913.
 „ primary, 926.
 „ secondary, 875, 927.
 Lathyrism, 280.
 Laveran's malarial parasite, 237.
 Lead, colic, 289, 291.
 „ "encephalopathy," 290.
 „ palsy, 291.
 Lead poisoning, 289.
 „ albuminuria, 291, 788.
 „ anæmia, 289, 290, 291.
 „ anuria, 780.
 „ aphasia, 290.
 „ blindness, 290.
 „ cachexia, 291.
 „ colic, 289, 290.
 „ constipation, 289.
 „ encephalopathy, 290, 291.
 „ epileptic fits, 290.
 „ gout, 290.
 „ gums, blue line, 290.
 „ kidney disease, 798.
 „ lumbar puncture, 291.
 „ nephritis, 798.
 „ nervous form, 290.
 „ neuro-muscular form, 290.
 „ oliguria, 290.
 „ sclerosis of cells of anterior horn, 944.
 „ urea, deficiency, 290.
 „ wrist drop, 290.
 „ leather bottle stomach, 643.
 Lenticulo-striate artery and apoplexy, 868.
 Leonine expression in leprosy, 125.
 Leprosy, cells, 125.
 „ maculo-anæsthetica, 124.
 „ tuberosa or tuberculosa, 124.
 Leprosy, 123.
 „ antivenene, 128.
 „ ageusia, 127.
 „ anosmia, 127.
 „ atrophy, 126.
 „ bacillus, 123.
 „ blindness, 125, 127.
 „ blood spread, 126.
 „ cells, 125.
 „ chaulmoogra oil, 128.
 „ degeneration, waxy, 127.
 „ eruptions, 126.
 „ fever, 124.
 „ fish, putrid, 124.
 „ forms, 124.
 „ geographical distribution, 123.
 „ heredity, 123.
 „ Hutchinson's view of, 124.
 „ iridocyclitis, 125.
 „ keratitis, 125.
 „ leonine face, 125.
 „ liver, 124.
 „ lymph spread, 125.
 „ maculo-anæsthetic, 124.
 „ nerves, 126.
 „ neuralgia, 125.

Leprosy—

- " spleen, 124.
- " testicle, 124.
- " tuberosa, 124.
- " tuberculous, 124.
- " ulceration, 125.

Leptomeningitis, acute, 849, 854.

- " encephalitis, 855.
- " primary formis, 849.
- " epidemic cerebro-spinal or meningococcal, 87, 853.
- " pneumococcal, 853.
- " tubercular, 850.
- " secondary, 854.
- " convulsions, 855.
- " brain abscess, 855.
- " middle ear disease, 855.
- " edema of membranes, 855.
- " chronic, 856.

Leucin, in phosphorus poisoning, 787.

- " urine, 787.
- " yellow atrophy of liver, 725.

Leucocytes, eosinophile, 342, 344, 347.

- " granular, 343.
- " hyaline, 343.
- " neutrophile, 342.

Leucocythæmia, 360 (*see* leukaemia).

Leucocytosis, 345.

- " appendicitis, 692.
- " cerebral abscess, 895.
- " gout, 319.
- " hæmoglobinuric fever, 247.
- " hepatic abscess, 712, 713.
- " absent in influenza, 346.
- " late Hodgkin's disease, 377.
- " absent in measles, 346.
- " myxœdema, 394.
- " pathological, 345.
- " physiological, 345.
- " post-hæmorrhagic anæmia, 346, 358.
- " leukaemia, spleno-medullary, 361.
- " pneumonia, 346, 537.
- " scurvy, 385.
- " scarlet fever, 169.
- " splenic anæmia, 370.
- " suppurations, 345.
- " absent in tuberculosis, 346.
- " absent in typhoid fever, 60, 68.

Leucomyelitis, 897.

Leucopenia, 346.

- " splenic anæmias, 346, 371.
- " pernicious anæmia, 355.

Leukaemia, 360.

- " adenoids, 495.
- " anæmia, 358.
- " apoplexy, 368.
- " asthenia, 369.
- " arsenic, 370.
- " bone marrow, 364, 367.
- " colour index, 351.
- " eosinophilia, 362, 364.
- " epistaxis, 368.
- " fever, 368.
- " gastric disturbance, 368.
- " hæmorrhages, 368.
- " heredity, 360.
- " hyperplasia, 364, 367.
- " kidney, 366.
- " liver, 366.
- " lymphocytes, 363.
- " lymphatic glands, 366, 368.
- " lymphatic form, 363.
- " mast cells, 363, 364.
- " myelocytes, 362, 364.
- " myelogenous form, 361.
- " normoblasts, 361.
- " polymorphonuclears, 362, 364.
- " priapism, 368.
- " pseudo, 370, 374.
- " retinitis, 368.
- " spleen, 365, 368.
- " thymus gland, 397.
- " uric acid excretion, 783.

Leukoplakia of mouth, 609.

Lice, 273.

Lichenous eruption after vaccination, 161.

Lienteric diarrhœa, 685.

Lightning pains in tabes dorsalis, 932.

Limmitzer's bacillus, 512.

Lipoma, 38.

Lipomatosis, general and local, 32.

Lipping of ends of bones in arthritis deformans, 309.

Lips, ulcers on in hereditary syphilis, 189.

Lithopædion, 33.

Lithæmia, 784, 810.

Little's disease, 924.

Liver, diseases of, abscess, 709, 711, 712.

- " alcoholic, 715.
- " aspiration, exploratory, 712.
- " atrophic, 715.
- " bactericidal action, 709, 714.
- " cancer, 728.
- " cirrhosis, 714.

Liver, diseases of—

- " congestions, 707.
- " degenerations, 707.
- " embolism, 708.
- " fibroses, 713-723.
- " gin drinker's, 715.
- " gumma, 719.
- " hobnailed, 715.
- " hydatids, 260, 729.
- " inflammations, 709, 726.
- " new growths, 727.
- " parasites, 729.
- " sarcoma, 728.
- " thrombosis, 708.
- " yellow atrophy, 724.
- Liver, cancer of, ascites in, 729.
- " " asthenia, 729.
- " " biliary, 728.
- " " cholæmia, 729.
- " " cirrhotic, 728.
- " " hematemeses, 729.
- " " hypernephritic, 728.
- " " jaundice, 279.
- " " massive, 728.
- " " secondary, 728.
- " " umbilication, 728.
- Liver, cirrhosis, 713.
- " " abdomen, tapping, 722, 723.
- " " alcohol, 714, 722.
- " " anastomoses, accessory venous, 717, 718.
- " " ascites, 715, 718, 720, 721.
- " " asthenia, 722.
- " " atrophic, 714.
- " " biliary, 715.
- " " bile ducts, new, 717.
- " " blood-vessels, new, 717.
- " " caput medusæ, 718.
- " " cancer, 728.
- " " coma, 720, 722.
- " " delirium, 720, 722.
- " " epistaxis, 720.
- " " facies, hepatic, 721.
- " " fatty, 714.
- " " fever, 720.
- " " gummata, 719.
- " " Hanot's, 715.
- " " hæmochromatosis, 752.
- " " hæmorrhoids, 718, 720.
- " " hæmatoporphyrinuria, 786.

Liver, cirrhosis—

- " " hematemesis, 720.
- " " hypertrophic, 715, 717.
- " " intercellular, 719.
- " " intestinal catarrh, 718.
- " " jaundice, 715, 722, 721.
- " " Laennec's, 715.
- " " leucin and tyrosin, 786.
- " " melæna, 720.
- " " monolobular, 714.
- " " pericellular, 714.
- " " peritonitis, chronic, 768.
- " " polylobular, 714.
- " " regeneration of hepatic cells, 716, 717.
- " " size of liver, 715, 720, 721.
- " " spleen, 718, 720, 721.
- " " symptoms, absent, 720.
- " " syphilitic, 718.
- " " hereditary, 719.
- " " toxæmia, 720.
- " " tubercle, 718, 722.
- " " urea diminution, 781.
- " " urobilinuria, 786.
- Liver, yellow atrophy, 724.
- " " abortion, 727.
- " " albuminuria, 726.
- " " angio-cholitis, 726.
- " " bacteria, 724.
- " " bile ducts, new, 725.
- " " coma, 726.
- " " delirium, 726.
- " " fever, 727.
- " " hæmorrhage, 725, 727.
- " " hematemeses, 726.
- " " jaundice, 726.
- " " liver, size, 724, 725.
- " " leucin, 725, 726.
- " " necrosis, 725.
- " " phosphorus poisoning, 724.
- " " pregnancy, 724.
- " " sex, 724.
- " " toxæmia, 725.
- " " tyrosin, 725, 726.
- " " urea diminution, 781.
- " " vomiting, 725.
- Living skeleton, 916.
- Lobar pneumonia, 534 (see pneumonia).
- " pseudo. 544.

- Lobular pneumonia, 513.
 " adults, 515, 557.
 " bacteria, 516.
 " children, 515, 559.
 " collapse, 514, 515.
 " diphtheria, 515.
 " influenza, 60, 515, 519.
 " measles, 515.
 " primary, 545.
 " secondary, 515.
 " sputum, 515.
 " small-pox, 152.
 Local peritonitis, 765.
 Lock-jaw, 103, 106.
 Locomotor ataxia, 929 (*see* tabes dorsalis).
 Loose shoulders in pseudo-hypertrophic paralysis, 923.
 Lordosis in pseudo-hypertrophic paralysis, 923.
 " progressive muscular atrophy, 916.
 Ludwig's angina, 615.
 Lumbago, 303, 304.
 Lumbar plexus, 1026.
 Lumbar puncture in epidemic cerebro-spinal meningitis, 79.
 " " fracture of skull, 880.
 " " hydrocephalus, 859, 860, 861.
 " " lead poisoning, 291.
 " " sleeping sickness, 252.
 " " spinal hæmorrhage, 800.
 " " tubercular meningitis, 853.
 Lung, abscess, 549.
 " bronchiectasis, 551.
 " bronchorrhœa, 550, 551.
 " empyema, 549, 557.
 " liver abscess, 550, 711.
 " mediastinal abscess, 550.
 " osteomyelitis, 549.
 " pneumothorax, 596.
 " subphrenic abscess, 550, 766.
 Lung, caseous tuberculosis, acute, 561.
 " " albuminuria, 569.
 " " bronchitis, 565.
 " " caseation, 565, 568.
 " " cavities, 566.
 " " cough (hacking), 569.
 " " cyanosis, 569.
 " " dyspnoea, 569.
 Lung, caseous tuberculosis—
 " " evolution of lesions, 567.
 " " fever, 569.
 " " hæmoptysis, 569.
 " " inœmia, 569.
 " " lobar pneumonia, 569.
 " " pleural changes, 565, 566.
 " " spleen, 569.
 " " sputum, 569.
 " " typhoid fever, 569.
 Lung, diseases of, abscess, 549.
 " actinomycosis, 131, 591.
 " apoplexy, 539, 531.
 " brown induration, 526.
 " collapse, 522.
 " congestion, passive, 446, 525.
 " hypostatic, 525.
 " emphysema, 518.
 " fever, 535.
 " gangrene, 552.
 " gumma, 590.
 " hæmorrhage, 528.
 " hydatids, 593.
 " inflammation, 532.
 " infarct, 11, 530.
 " œdema, 527.
 " syphilis, 590.
 " tuberculosis, 561.
 " tumors, 591.
 Lung fever, 535.
 Lung, miliary tuberculosis, acute, 561.
 " " bronchitis, 561.
 " " bronchiectasis in children, 512.
 " " cerebral symptoms, 563.
 " " choroid miliary tubercles, 563.
 " " chronic, 562.
 " " cyanosis, 563.
 " " dyspnoea, 563.
 " " emphysema in children, 561.
 " " fever, 563.
 " " hæmogenic form, 516.
 " " lymphogenic form, 561.
 " " meninges, 563.
 " " spleen, 562, 576.
 " " sputum, 563.
 " " thoracic duct infection, 562.

- Lung, miliary tuberculosis—
 " " toxæmia, 563
 " " typhoid state, 563.
 Luy's separator, 797, 820.
 Lymphatic glands, in anthrax, 223
 " chloroma, 372.
 " diphtheria, 95, 97
 " erysipelas, 140.
 " exophthalmic gout, 397.
 " filariasis, 270.
 " gastric cancer, 664.
 " glanders, 220.
 " Hodgkin's disease, 375, 377.
 " leprosy, 125.
 " leukæmia, 366, 368.
 " Malta fever, 88.
 " œsophageal diverticulum,
 624.
 " pyæmia, 137.
 " rheumatic arthritis, 310.
 " scarlet fever, 167.
 " sleeping sickness, 252.
 " splenic anæmia, 370.
 " syphilis, 185.
 " tuberculosis, 111, 115 ‡
 " typhoid fever, 65.
 " waxy disease, 30.
 Lymphadenoma, 374.
 " lymphæmia, 367, 375, 378
 " lymphosarcoma, 367, 375,
 378.
 Lymphæmia, 363
 " lymphosarcoma, 367.
 Lymphocytosis, 346.
 " anæmia, pernicious, 355.
 " chloroma, 372.
 " some cases of Hodgkin's
 disease, 377.
 " infancy, 346.
 " lymphatic leukæmia, 363.
 " measles, 358.
 " rickets, 346, 358.
 " sleeping sickness, 252.
 Lymphorrhagia, 271.
 Lymphosarcoma and chloroma,
 373
 " lymphæmia, 367.
 Lymph, scrotum, 270.
 " vaccine, 159.
 " varix, 270
 Lysis in typhoid fever, 62.
 Lysophobia, 230.
 Macrophages, 19
 Macular skin eruption in secondary
 syphilis, 186.
 Madura foot, 132.
 Main en griffe, 916.
 Major epilepsy, 971.
 Malaria, 235.
 " anæmia, 242, 245
 " arsenic, 245.
 " benign, 238.
 " bilious remittent, 244.
 " cold stage, 243.
 " crescents, 240, 244.
 " fever, 238, 243
 " Grassi's experiments, 242.
 " hæmoglobinuric fever, 246.
 " hæmoglobinuria, 791.
 " hot stage, 243.
 " immunity, 237.
 " intermittent, 242, 243
 " jaundice, 741.
 " latent phase, 244.
 " Laveran's plasmodium, 237.
 " liver, 242.
 " long intervals, 244.
 " malignant, 238, 240.
 " micro-organism, 237.
 " mosquito (anopheles), 240,
 246.
 " nephritis, 799, 806.
 " pernicious, 242, 244.
 " pigment, 237, 242, 243
 " quartan, 238, 239.
 " quinine, 245.
 " quotidian, 238, 239.
 " remittent, 242, 244.
 " race incidence, 237.
 " reproduction, asexual, 238.
 " " sexual, 241.
 " rigors, 243, 244.
 " Sambon and Low's experi-
 ments, 242.
 " seasonal variations, 236,
 238.
 " spleen, 242, 245.
 " sporulation, 238, 239, 240.
 " sweating, 243.
 " tertian, 238, 239.
 " typho-malarial, 244.
 " uric acid, excess, 783.
 Malignant endocarditis types, 410
 Malignant pustule, 222, 223.
 Mallei bacillus, 219
 Mallein, 221.
 Malta fever, 87.
 " anæmia, 88.
 " arthritis, 88.
 " fever, 88.
 " geographical distribution, 87.
 " goats' milk as carrier of
 infection 87.

Malta fever—

- " incubation period, 87.
- " loss of hair, 88.
- " lymphatic glands, 88.
- " micrococcus, 87.
- " orchitis, 88.
- " relapses, 88.
- " spleen, 87, 88.

Mandl's solution in chronic tonsillitis, 623.

Marriage, question of, in epilepsy, 975.

- " syphilis, 184.
- " tuberculosis, 121, 583.

Massive pneumonia, 539.

Mast cells, 344.

Mastication spasm of muscles, 1047.

M'Burney's point, 691:

Measles, 172.

- " age, 172.
- " albuminuria, 788.
- " bronchitis, 173, 505.
- " broncho-pneumonia, 174.
- " cancrum oris, 613.
- " contagiousness, 172.
- " convulsions, 174, 978.
- " coryza, 173.
- " desquamation, 173.
- " diarrhoea, 173.
- " fever, 173.
- " gangrene, 173.
- " German, 175.
- " immunity, 173.
- " incubation period, 173.
- " nephritis, 798, 799, 802.
- " otitis media, 174.
- " pneumonia, lobular, 545.
- " rash, mulberry-like and crescentic, 173.
- " mouth, 172.
- " whooping-cough, 174.

Measley pork, 257.

Meat pies, poisoning, 279.

Meckel's diverticulum, 672.

Median nerve paralysis, 1025.

Mediastino pericarditis, indurative, 416.

Mediastinum actinomycosis, 130.

- " malignant disease, 431.

Mediterranean fever, 87.

Medulla oblongata, lesion, 955.

Megaloblasts, 342.

Megalocytes, 342.

Melena, 658.

- " duodenal ulcer, 660.
- " gastric ulcer, 658.
- " hepatic cirrhosis, 720.

Melanin pigment, 35.

Melanotic sarcoma, 35.

Melanuria, 793.

Melitensis micrococcus, 87.

Membranous croup, 96, 102.

Meniere's disease, 1056.

- " tinnitus aurium, 1057.

- " vertigo, 1057.

- " vomiting, 1057.

Meningeal hæmorrhage, 844, 867, 879.

Meninges, 844.

Meningitis, acute cerebro-spinal, 849.

- " epidemic cerebro-spinal, 77.

- " lepto-, 849.

- " meningococcal, 853.

- " pachy-, 845.

- " pneumococcal, 853.

- " posterior basal, 77.

- " primary, 849.

- " sporadic, 77.

- " tubercular, 850 (*see* leptomeningitis).

Meningitis, tubercular, 850.

- " " abdomen, contracted 852.

- " " brain compression, 852.

- " " caseous tubercle, 851.

- " " coma, 852.

- " " choroid tubercles, 852.

- " " convulsions, 852.

- " " delirium, 852.

- " " encephalitis, 851, 855.

- " " exudate, 850, 854.

- " " headache, 852.

- " " hydrocephalic cry, 852.

- " " hydrocephalus, internal, 851.

- " " Jacksonian attacks, 852.

- " " Kernig's sign, 852.

- " " leucocytosis, 853.

- " " lumbar puncture, 853.

- " " miliary tubercle, 850.

- " " neuritis (optic), 852.

- " " paralyzes, 852.

- " " primary (clinically), 850.

- " " pulse, 852.

- " " pupils, 852.

- " " rigors, 852.

Meningitis, tubercular—
 " " secondary, 850.
 " " squint, 852.
 " " tache cerebral, 853.
 " " vomiting, 852.
 Meningo-encephalitis, 855.
 Meningo-myelitis, 897.
 Menorrhagia, 12.
 Mercurial tremor, 1017.
 Merycismus, 670.
 Mesenteritis, 475.
 Mesenteric artery, superior, embolism, 678.
 Mesenteric glands, tuberculosis, 116.
 Metallic, poisoning, 287.
 " rales in lung cavities, 580.
 Metastatic, abscesses, 137.
 " tumors, 38.
 Metatarsal neuralgia, 1008.
 Meteorism, in peritonitis, 762.
 " typhoid fever, 65, 72.
 Methæmoglobin, 388, 389.
 Metrorrhagia, 12.
 Microcytes, 342.
 Micro-organism in actinomycosis, 129.
 " angio-cholitis, suppurative, 742.
 " appendicitis, 688.
 " atrophy, acute yellow, 724.
 " beri-beri, 211.
 " bronchitis (acute), 503.
 " bronchiectasis, 513.
 " cancrum oris, 612.
 " cholangitis, suppurative, 742.
 " cholecystitis, 737.
 " coccidiosis, 248, 249.
 " dengue, 206.
 " diabetes mellitus, 329.
 " dysentery, 208.
 " degeneration, waxy, 31.
 " endocarditis, acute, 433, 434.
 " benign, 436.
 " malignant, 439.
 " enteritis, general, 681.
 " erysipelas, 139.
 " food poisoning, 279.
 " foot and mouth disease, 234.
 " gall stones, 730.
 " gangrene, 21.
 " gastritis niembranous, 650.
 " " phlegmonous, 649.
 " gonorrhœal rheumatism, 305.
 " hæmoglobinuric fever, 246, 247.

Micro-organism—
 " hepatic abscess, 711.
 " " cirrhosis, 714.
 " " suppurative, 711.
 " hepatitis, acute, 709.
 " Hodgkin's disease, 374.
 " hydrophobia, 226, 229.
 " laryngitis, catarrhal, 496.
 " " niembranous, 102.
 " leukæmia, 360.
 " lung abscess, 549.
 " " gangrene, 552.
 " " inflammations, 533.
 " malignant tumors, 39, 249.
 " madura foot, 132.
 " malaria, 237.
 " meningitis, epidemic cerebro-spinal, 77.
 " " primary, 848.
 " " secondary, 853.
 " molluscum contagiosum, 249.
 " mouth, putrid sore, 611.
 " mycetoma, 132.
 " nasal catarrh, 488.
 " necrosis, 20.
 " nephritis, 800.
 " otitis media of influenza, 91.
 " Paget's disease of nipple, 249.
 " pancreatitis, acute, 749, 750.
 " pericarditis, acute, 411.
 " peritonitis, 759.
 " pleurisy, acute, 598, 599.
 " pneumonia, chronic, 556.
 " " lobar, 535.
 " lobular, 546.
 " pneumonia, 549.
 " pneumothorax, 596.
 " psorospermiasis, 248, 249.
 " ptomaine poisoning, 279.
 " pyæmia, 137.
 " relapsing fever, 52.
 " rheumatic arthritis, chronic, 308.
 " " fever, 294.
 " scarlet fever, 163.
 " septicæmia, 135.
 " stomach, healthy fasting, 631.
 " stomatitis, aphthous, 609.
 " " catarrhal, 603.
 " " parasitic, 610.
 " ulcerative, 611.
 " syphilis, 183.
 " thrush, 610.

Micro-organism—

- „ typhus fever, 193.
- „ yellow fever, 202.

Microphages, 19.

Middle Cerebral Arteries, embolism, 884.

- „ thrombosis, 884.

Migraine, 983.

- „ nerve storm theory, 984.
- „ vision disturbances, 984.

Miliary aneurisms, 869.

- „ gummata, 719.
- „ tubercle, 114.

Milk and cholera, 81.

- „ diphtheria, 93.
- „ enteritis, 681.
- „ Malta fever, 87.
- „ scarlet fever, 163.
- „ typhoid fever, 59.
- „ tuberculosis, 59, 113

Milk spots on heart, 413.

Mind, blindness, 961.

- „ deafness, 962.

Miner's anæmia from ankylostomiasis, 265.

- „ lung, 555.
- „ nystagmus, 1043.

Minor epilepsy, 973.

Miscarriage, 190.

Mitral incompetence, 445.

- „ „ albuminuria, 447.
- „ „ clubbing of fingers, 447.
- „ „ compensation, 446, 448.
- „ „ circulation, 447.
- „ „ death, sudden, 448.
- „ „ dropsy, 447, 448.
- „ „ dyspnœa, 447, 448.
- „ „ effect on heart, 446.
- „ „ expectation of life, 448.
- „ „ functional, 446.
- „ „ hæmaturia, 447.
- „ „ murmur, systolic mitral, 447.
- „ „ organic, 446.
- „ „ relative, 446.
- „ „ second sound accentuated, 447.
- „ „ sputum, hæmorrhagic, 448.

Mitral stenosis, 449.

- „ „ age, 449.
- „ „ hæmoptysis, 450.
- „ „ murmur, diastolic mitral, 450.

Mitral stenosis—

- „ „ presystolic, 450.
- „ „ pulse, 450.
- „ „ second sound, accentuated and reduplicated, 405.
- „ „ thrill, presystolic, 450
- „ „ thrombi, 449.

Mitral valve, button hole, 444.

- „ „ funnel shaped, 443.
- „ „ incompetence, 445.
- „ „ rheumatic fever, 431, 445.

„ „ stenosis, 449.

Moles, pigmented, 35.

Mollities ossium, 1003.

Monoplegia, cerebral, 838.

- „ hysterical, 988.

Morbili, 172.

Morbus maculosus of Werlhof, 383.

Morphia habit, 283.

Morphinism, 283.

Mortality from respiratory disease, 488.

Morton's disease, 1008.

Morvan's disease, 864.

- „ leprosy, 864.

Mosquitoes as carriers of filaria, 270.

- „ malaria, 241.

- „ yellow fever, 202.

Motor system, 834.

Mountain ascents and epistaxis, 494.

Mouth breathing in adenoids, 495.

- „ „ nasal catarrh, chronic, 492.

Mouth diseases, 608.

- „ inflammations, 609-614.

- „ putrid sore, 611.

Movable kidney, 772 (*see* kidney).

Mucous colitis, 684.

Mucous patches, 186.

Mulberry rash in typhus fever, 195.

Multiple gangrene, 997.

Multiple cerebro-spinal sclerosis, 948.

Mummification, 21.

Mumps, 181, 615.

Murmurs, in anæmia, pernicious, 356.

- „ aneurism, 485.
- „ chlorosis, 351.
- „ Flint's, 453.
- „ functional cardiac, 428.
- „ postural effect, 450.
- „ valvular diseases, 437, 441, 447, 450, 452, 455.

Muscarin, 280.

Muscles, diseases, 1068

Muscular atrophies, varieties, 913

- „ adults, 921.
- „ amyotrophic, 914
- „ Aran-Duchenne, 914
- „ atonic, 916.
- „ bulbar, 914, 917.
- „ central, 913.
- „ children, 921.
- „ facio-scapulo-humeral, 921.
- „ peroneal, 920.
- „ progressive, 913
- „ spinal, 913
- „ tonic, 916.

Muscular contractures in hemiplegia, 875

- „ hysteria, 988.
- „ progressive muscular atrophy, 916.

Muscular cramps in beri-beri, 212.

- „ cholera, 83.
- „ uræmia, 782.

Muscular dystrophies, 921.

Muscular rheumatism, 303

Muscular sense, in tabes dorsalis, 933.

Musculo-spiral paralysis, 1024

Mushroom poisoning, 280.

- „ like cholera, 85

Musical murmur of mitral incompetency, 447.

Myasthenia gravis, 982

Myasthenic reaction, 982

Mycetoma, 132.

- „ colonies, black and white, of germs, 132.
- „ mycelium, 132.
- „ resemblance to actinomycosis, 132

Mycotic gastritis, 649.

Myelæmia, 361.

Myelitis, 897.

- „ acute, 897, 901, 902.
- „ central, 897.
- „ cervical, 901.
- „ chronic, 897, 903, 912.
- „ compression, 899, 903
- „ degenerations, secondary, 898.
- „ diffuse, 897, 901, 904
- „ disseminated, 897.
- „ hæmorrhages, 898.
- „ infections, 897, 899.
- „ leucomyelitis, 897.
- „ localised, 901
- „ lymphogenic forms, 898.
- „ meningomyelitis, 897.

Myelitis—

- „ poliomyelitis, 897, 904, 913
- „ sclerosis, 913.
- „ suppurative, 898.
- „ toxic, 898
- „ transverse, 897, 899, 901.
- „ traumatic, 897.

Myelitis, diffuse, 901.

- „ arthritis, 902.
- „ bedsores, 902.
- „ motor paralysis, 902.
- „ reflexes, 902

Myelitis, transverse, 901.

- „ bedsores, 901.
- „ girdle sensation, 901.
- „ hyperæsthesia, 901.
- „ muscular atrophy, contractures, paralysis and spasticity, 901.
- „ reflexes, 901.

Myelocyte, 344.

Myelocythæmia, 361.

Myelogenous leukaemia, 361

Myocarditis, acute, 421.

- „ chronic, 119, 422.
- „ fibrous, 419, 422
- „ suppurative, 422
- „ syphilitic, 423
- „ tubercular, 423

Myocardium, diseases of, abscess, 421

- „ atrophy, 421
- „ degeneration, fatty, 417.
- „ „ fibroid, 419
- „ „ waxy, 119
- „ fragmentation and segmentation, 420
- „ infiltration, fatty, 418
- „ inflammation, 421, 422.
- „ softening, 424
- „ thrush breast appearance, 417
- „ tumors, 431

Myoclonia, 982

Myomalacia cordis, 421

Myosis, spinal, 934.

Myositis, 1068

- „ ossificans progressiva, 1068

Myotonia congenita, 1069

Myotonic reaction of Erb, 1070

Myœdema 393

- „ facies, 394.
- „ hæmorrhage, 394
- „ hair, loss, 394.
- „ leucocytosis, 394
- „ sex, 395
- „ speech, 394

Myxœdema—

- thyroid gland, 394.
 " " extract, 395.

Nagana, 251.

Nasal catarrh, acute, 488.

- chronic, 490.
 " deafness, 491.

Naso-pharynx resonance, interference with, 495.

Nauheim treatment of chronic bronchitis, 509.

- heart disease, 420.

Neapolitan fever, 87.

Neck cellulitis, 615.

Necrosis, 20.

Negri bodies in rabies, 230.

Nematodes, 261.

Nephrectomy, 822.

Nephritis, 798.

- acute, 799.
 acute interstitial, 802.
 alcohol, 798, 806.
 cantharides, 799.
 chill, 800, 804.
 cholera, 800.
 chronic, 804, 806.
 diphtheria, 96, 98, 799, 802.
 glomerular, 799, 807.
 glottidis œdema, 803.
 gout, 319, 322, 798.
 interstitial, 799.
 lead, 798.
 measles, 799, 802.
 parenchymatous, 799.
 pneumonia, 537, 540, 800.
 pregnancy, 800.
 scarlet fever, 168, 171, 799, 800, 802.
 small-pox, 800.
 syphilis, 799, 800, 806, 818.
 tonsillitis, 800.
 transient, 798, 801.
 tubercle, 800, 806, 818.
 typhoid fever, 800.
 vinylamine, 799.

Nephritis acute, 799.

- albuminuria, 788, 803.
 anæmia, 804, 806.
 casts, 802, 803.
 causes, 799.
 cardiac dilatation, 804.
 children, 802.
 convulsions, 803.
 death, sudden, 804.

Nephritis acute—

- diet, 805.
 dropsy, 803.
 fatty degeneration, 801.
 gastro-intestinal symptoms, 804.
 glomerular changes, 802.
 hæmorrhages, 801.
 hæmaturia, 803.
 interstitial changes, 802.
 prognosis, 804.
 retinitis, 804.
 tubular changes, 802.
 urea diminution, 803.
 urine, diminished, 803.
 uræmia, 804, 805.

Nephritis chronic interstitial, 809.

- age, 811.
 albuminuria, 815.
 albumosuria, 815.
 amaurosis (uræmic), 816.
 anæmia, 816.
 arterio-sclerosis, 477, 810, 814.
 bronchitis, 814.
 cardiac hypertrophy, 426, 814, 815.
 causes, 810, 811.
 cysts, 825.
 diarrhœa, 816.
 dropsy, 815.
 fat, 812.
 glomerular changes, 813.
 glottidis œdema, 498, 803, 815.
 hæmorrhages, 814.
 cerebral, 814, 816.
 gastric, 814.
 nasal, 814, 816.
 retinal (flame-shaped), 814, 816.
 heredity, 812.
 interstitial changes, 813.
 latency, 814.
 meningitis, 855.
 pericarditis, 814.
 pleurisy, 814.
 pneumonia, 814.
 pulse (hard), 815.
 retinitis, 816.

Nephritis chronic interstitial—

- " " se\, 811.
- " " tubular changes, 813.
- " " uræmia, 814, 815, 816, 817.
- " " urea, amount, 815.
- " " urine, amount and specific gravity, 815.
- " " vomiting, 816.

Nephritis chronic parenchymatous, 806.

- " " albuminuria, 808.
- " " anæmia, 808.
- " " casts, 807, 808.
- " " cardiac hypertrophy, 808.
- " " causes, 806.
- " " complexion, earthy, 808.
- " " cysts, 808.
- " " death, sudden, 809.
- " " diarrhoea, 808.
- " " dropsy, 808, 809.
- " " glomerular changes, 807.
- " " hæmaturia, 808.
- " " hæmorrhagic form, 806.
- " " hæmorrhage cerebral, 808.
- " " interstitial changes, 807.
- " " kidney, white, 807.
- " " granular, 807.
- " " pigmentation, skin, 808.
- " " tubular changes, 807.
- " " uræmia, 808.
- " " urea, diminished, 808.
- " " urine, amount, 808.

Nephrolithiasis, 795.

Nephrorraphy in movable kidney, 773.

" Raynaud's disease, 998.

Nephrotomy, 822.

Nerves, diseases of, 1004.

- " cranial, 1028.
- " inflammation, 1012.
- " pain, 1004.
- " spinal, 1020.
- " tumors, 1019.

Nerves, lesions of, anterior crural, 1026.

- " auditory, 1054.
- " brachial plexus, 1022.

Nerves, lesions of—

- " chorda tympani, 1051.
- " circumflex, 1024.
- " cochlear, 1054.
- " eighth, 1034.
- " external cutaneous, 1026.
- " facial, 1048.
- " fifth, 1043.
- " fourth, 1039.
- " glossopharyngeal, 1058.
- " hypoglossal, 1066.
- " intermedius of Wrisberg, 1047.
- " long thoracic, 1024.
- " lumbar plexus, 1026.
- " median, 1025.
- " musculo-spiral, 1024.
- " obturator, 1026.
- " olfactory, 1028.
- " optic, 1029.
- " phrenic, 1020.
- " recurrent laryngeal, 1060.
- " sciatic, 1027.
- " sixth, 1040.
- " spinal accessory, 1063.
- " third, 1036.
- " ulnar, 1025.
- " vagus, 1059.
- " vestibular, 1056.

Nerve roots, lesions, 1023.

" Nerve storm," theory of Migraine, 983.

Nerve stretching in sciatica, 1011.

Nervous system, diseases of, 831.

- " membranes, 844.
- " substance of brain and cord, 857.
- " functional, 962.
- " vasomotor and trophic, 995.
- " nerves, 1004.

Netter's statistics on pneumococcus in lung lesions, 536.

Neuralgia, 1004.

- " brachial plexus, 1006.
- " breast, 1006.
- " causes, 1004.
- " coccygeal, 1008.
- " intercostal, 1006.
- " leprosy, 126.
- " lumbar plexus, 1006.
- " metatarsal, 1008.
- " ovary, 1008.
- " paroxysmal, 1006.
- " pelvic, 1008.
- " periodicity, 1005.
- " sciatica, 1007.
- " spinal nerves, 1006.

Neuralgia—

- " supraorbital, 1015.
- " testicle, 1008.
- " trigeminal, 1005.
- " visceral, 1008.

Neurasthenia, 991.

- " agoraphobia, 993.
- " anthropophobia, 993.
- " insomnia, 993, 994.
- " mental symptoms, 993.
- " traumatic, 994.

Neuritis, 1012.

- " alcoholic, 1013, 1016.
- " arsenical, 1017.
- " ascending, 1014.
- " cold, 1013.
- " interstitial, 1012.
- " lead poisoning, 1017.
- " local, 1012.
- " migrans, 1014.
- " multiple, 1015.
- " optic, 1031.
- " parenchymatous, 1012.
- " peripheral, 1015.
- " rheumatism, 1013.
- " sympathetic, 1014.

Neuritis, multiple, 1015.

- " alcohol, 1015, 1016.
- " anæmia, 1015.
- " arsenical, 1017.
- " beri-beri, 1015.
- " cancer, 1015.
- " diabetes mellitus, 1015.
- " diphtheria, 98, 1015.
- " lead, 1017.
- " mercury, 1017.
- " leprosy, 1015.
- " syphilis, 1015.

Neuritis, peripheral alcoholic, 1016.

- " electrical reactions, 1017.
- " foot drop, 1016.
- " hallucinations, 1017.
- " high steppage gait, 1016.
- " motor changes, 1016.
- " numbness, 1016.
- " pain in muscles, 1016.
- " reflexes usually lost, 1017.
- " trophic changes, 1017.
- " wrist drop, 1016.

Neuritis, peripheral, from lead, 1017.

- " types of paralysis in arm, 1018.
- " hand, 1018.
- " laryngeal, 1018.
- " peroneal, 1018.
- " foot drop, 1018.
- " wrist drop, 1017.

Neurofibromata multiple, 1019.**Neuromata, 1019.**

- " amputation, 1020.
- " false, 1019.
- " multiple, 1020.
- " plexiform, 1019.

Neurone, 831.

- " arborisations, 832.
- " axon, 832.
- " chromatolysis, 833.
- " decussation, 836, 842.
- " dendrites, 832.
- " motor, 834-841.
- " Nissl's granules, 832.
- " regeneration, 833.
- " reaction a distance, 834.
- " sensory, 841-844.
- " synapse, 832.
- " systems, 834.

Neuroses, occupation, 978.**Neutrophile leucocytes, 342.****New growths, 37.****Night blindness, 1035.****" scurvy, 385.****Night sweats in phthisis, 579.****" treatment, 589.****Ninth nerve lesions, 1058.****Nissl bodies (tigroid), 832.****Nits, 273.****Nodes, Heberden's, 310.****Noma, 612.****Non-synchronous action of auricles and ventricles, 463.****Normoblasts, 342.****Nose, bleeding, 493.****" diseases, 488.****Nucleins and gout, 319.****Nummular sputum in chronic phthisis, 577.****Nutmeg liver, 4.****Nutritional changes in fever, 44.****Nyctalopia, 1035.****" scurvy, 385.****Nystagmus, 1042.****" cerebellar lesions, 943, 956.****" Friedreich's ataxia, 942.****" hydrocephalus, 860.****" miners', 1043.****" multiple cerebro-spinal sclerosis, 950.****" ocular causes, 1043.****" rickets, 327.****" trade, 1043.****Obstruction of bowel, 672.****" acute, treatment, 676.****" chronic, treatment, 677.**

- Obturator nerve, lesions, 1026.
 Occipital lobe, lesions, 954.
 Occupation neuroses, 979.
 Ochlotic fever, 192.
 Ochronosis, 793.
 Ocular muscles, paralysis, 1041.
 Ocular muscles, spasms, 1042.
 " epilepsy, 1042.
 " hysteria, 1042.
 " nystagmus, 1042.
 " squint, 1042.
 Edema, 5.
 " angio-neurotic, 995.
 " brain, 867.
 " erysipelatous in anthrax, 224.
 " glottidis, 497.
 " lungs, 527.
 Esophageal veins in hepatic cirrhosis, 626, 720.
 Esophagismus, 625.
 Esophagitis, acute, 626.
 " chronic, 626.
 " phlegmonous, 626.
 Esophagus, diseases, 624.
 " cancer, 627.
 " diverticulum, 624.
 " foreign bodies, 625.
 " inflammation, 626.
 " stenosis, 625.
 " ulceration, in syphilis, 187.
 Esophagus, cancer, 627.
 " asthenia, 628.
 " cervical glands, 628.
 " emaciation, 627.
 " regurgitation of food, 627.
 Esophagus, stenosis of, from aneurism, 625.
 " cancer, 627.
 " foreign bodies, 625.
 " spasmodic, 625.
 Oidium albicans, 610.
 Olfactory nerve, lesions, 1028.
 " anosmia, 1029.
 " hyperosmia, 1029.
 " parosmia, 1029.
 Oliguria in lead poisoning, 290.
 Omentum, hydrops, 756.
 " cancer, abdominal, 664.
 " suppuration, 690.
 Ophthalmoplegia, 1041.
 " external, 1041.
 " internal, 1041.
 " syphilis, 1041.
 Opisthotonos, epidemic meningitis, 78.
 " tetanus, 107.
 Opium poisoning, 283.
 " coma, 283.
 " cyanosis, 283.
 " dyspnoea, 283.
 " facies, 284.
 " pupils, 283.
 " withdrawal of drug, 284.
 Opsonic method, in treatment, 587.
 Optic atrophy, 1033.
 " consecutive, 1033.
 " grey, 1033.
 " primary, 1033.
 " secondary, 1034.
 " " grey in tabes dorsalis, 934, 1033.
 " white, 1033, 1034.
 Optic chiasma and tracts, lesions, 1035.
 " heteronymous hemianopia, 1036.
 " nasal hemianopia, 1036.
 " Wernicke's pupil reaction, 1035, 1036.
 Optic neuritis, 1031.
 " brain abscess, 896, 1031.
 " aneurism, 1031.
 " cyst, 1031.
 " tumor, 952.
 " cerebellum and corpora quadrigemina, tumors, 952, 953, 1031.
 " pons and medulla (rarely), 953, 1031.
 " chronic Bright, 1031, 1034.
 " lead poisoning, 1031.
 " meningitis (tubercular), 852, 1031.
 " Leber's toxic theory, 1031.
 " peripheral scotoma, 1032.
 " trephining, 1032.
 Orchitis, in mumps, 181.
 " small-pox, 152.
 Orthotoncs in epidemic meningitis, 78.
 Osteitis deformans, 1003.
 Osteo-arthritis, hypertrophic pulmonary, 1002.
 Osteomalacia, 1003.
 Osteophytes in rheumatoid arthritis, 309.
 Otitis media, in brain abscess, 893.
 " meningitis, 855.
 " optic neuritis, 896.
 " scarlet fever, 168, 171.
 Ovaries, inflammation of, in mumps, 181.
 " small-pox, 152.

Oxalate of lime calculus, 796.

Oxaluria, 785.

„ lithæmia, 784.

„ stomach, dilatation, 795.

Oxygen inhalations in lobar pneumonia, 542.

Oxyuris vermicularis, 262.

Oysters and typhoid fever, 59.

Pachydermia laryngis, 499.

Pachymeningitis, 845.

„ cerebral, 845.

„ cervicalis hypertrophica, 847.

„ „ claw hand, 847.

„ „ head retraction, 864.

„ „ paraplegia, spastic, 847.

„ „ sensory symptoms, 847.

„ „ syringomyelia, 847.

„ „ tumors, 847.

„ externa, 845.

„ „ middle ear disease, 845.

„ „ sinistral thrombosis, 845.

„ interna, 845.

„ interna hæmorrhagica, 846.

„ dementia, senile, 846.

„ spinal, 846.

„ syphilis, 845, 846.

„ vertebral tuberculosis, 845.

Paget's disease, 1002.

„ nipple, 249.

Pain, in appendicitis, 691.

„ cancer of stomach, 665.

„ colic, appendical, 735.

„ „ biliary, 734, 735.

„ „ intestinal, 705.

„ „ renal, 735.

„ pleurisy, 602.

„ pneumonia, 583.

„ tetanus, 107.

„ ulcer of stomach, 658.

Palate, paralysis of, in diphtheria, 98.

„ lesion of vagus, 106c.

Palpation of kidney, 772.

„ pancreas, 753.

Palpitation, 463.

Palsies, birth, 924.

Palsy, lead, 290, 1017.

„ shaking, 962.

Pancreas, cancer of, diabetes mellitus, 753.

„ „ emaciation, 753.

Pancreas, cancer of—

„ „ jaundice, 753.

„ „ pain, 752.

Pancreas, diseases of, abscess, 751.

„ calculi, 754.

„ cancer, 752.

„ cysts, 753.

„ diabetes, 329, 747, 752.

„ digestion, 746, 747.

„ fat necrosis in diseases, 21, 750.

„ fibrosis, 752, 754.

„ gangrene, 751.

„ hæmorrhage, 748.

„ inflammations, 749.

„ secretion, internal, 747.

„ tumors, 752.

Pancreatitis, acute, 749.

„ chronic, 751.

„ fat necrosis, 750.

„ gangrenous, 751.

„ hæmorrhagic, 749.

„ peritonitis, 758.

„ subphrenic abscess, 765, 766.

„ suppurative, 750.

Panophthalmitis in inflammation of

Gasserian ganglion, 1045.

Papillitis, 1031.

Paracentesis pericardii, 415.

„ thoracis, 604.

Paræsthesia, 843, 933.

Parageusia, 1048.

Paralysis, abductor of larynx, 1061.

„ acute ascending, 910.

„ adductor of larynx, 1061.

„ agitans, 962.

„ atrophic, spinal, 904.

„ Bell's, 1050.

„ brachial plexus, 1022.

„ Brown-Séquard's, 843.

„ bulbar, 917.

„ „ acute, 918.

„ „ chronic, 918.

„ Caisson disease, 883.

„ cerebral compression, 845.

„ „ hæmorrhage, 872.

„ circumflex nerve, 1024.

„ ciliary muscle, 1039.

„ crutch, 1024.

„ diaphragm, 1021.

„ diphtheria, 98.

„ diver's, 882.

„ eleventh nerve, 1063.

„ Erb's, 1023.

„ Erb's juvenile, 921.

„ external popliteal, 1027.

„ facio-scapulo-humeral, 921.

Paralysis—

- „ fourth nerve, 1039.
- „ general, of insane, 944.
- „ glosso - labio - pharyngeal, 917.
- „ hysteria, 988.
- „ infantile, 904.
- „ internal popliteal nerve, 1027.
- „ Klumpke's, 1023.
- „ Landouzy-Dejerine's, 921.
- „ lead, 1017.
- „ long thoracic nerve, 1024.
- „ lower arm type, 907, 1023.
- „ median nerve, 1025.
- „ meningitis, tubercular, 852.
- „ muscles of mastication, 1046.
- „ musculo-spiral nerve, 1024.
- „ ninth nerve, 1058.
- „ obturator nerve, 1026.
- „ ocular muscles, 1041.
- „ œsophageal branch of vagus, 1062.
- „ phrenic nerve, 1020.
- „ primary spastic, of adults, 926.
- „ progressive, general, 944.
- „ pseudo-hypertrophic, 921.
- „ recurrent laryngeal, 1061.
- „ secondary spastic, 927.
- „ serratus, 1024.
- „ sixth nerve, 1040.
- „ syphilitic, spinal, of Erb, 927.
- „ third nerve, 1037.
- „ ulnar nerve, 1025.
- „ upper arm type, 907, 1023.
- „ uræmia, 783.

Paralysis agitans, 962.

- „ age, 962.
- „ attitude, 964.
- „ festinant gait, 964.
- „ rigidity, 964.
- „ tremor, 963.

Paramyoclonus multiplex, 982.

Paraphasia, 960.

Paraplegia, ataxic, 940.

- „ birth palsies, 925.
- „ compression myelitis, 900.
- „ hysteria, 927, 988.
- „ pachymeningitis cervicalis hypertrophica, 847.
- „ primary spastic paralysis of adults, 926.

Parasites, animal, 254.

- „ heart, 431.

Parasites—

- „ liver, 729.
- „ lung, 592.
- „ peritoneum, 729.
- „ pleura, 607.
- Parasitic stomatitis, 610.
- Parathyroids, 391.
- „ excision, 391.
- Parenchymatous nephritis, 799.
- Parieto-occipital lobe, lesions, 954.
- Parkinson's disease, 962.
- Parosmia, 1029.
- Parotitis, epidemic, 181, 615.
- „ age, 182.
- „ fever, 181.
- „ oophoritis, 181.
- „ orchitis, 181.
- „ pneumonia, 540.
- „ relapsing fever, 54.
- „ small-pox, 151.
- „ symptomatic in typhoid fever, 66, 615.
- Parotitis, symptomatic, 615.
- „ abdominal injuries, 615.
- „ typhoid fever, 66, 615.
- Paroxysmal hæmoglobinuria, 388, 791.
- „ tachycardia, 465.
- Patellar tendon reflex, 840.
- Pavy's test for sugar, 332.
- Pectoriloquy, 580.
- Pediculi, 273.
- Pedicular capitis, 273.
- „ vestimentorum, 273.
- Peliosis, rheumatic, 382.
- „ acute endocarditis, 435.
- Pellagra, 280.
- „ Raynaud's disease, 280.
- „ sclerosis of cord, 943.
- Pelvis, flat, in rickets, 315.
- Pelvis of kidney, calculi, 797.
- „ hæmorrhage from in granular kidney, 814.
- „ inflammation, 820.
- Peptic ulcer, 653.
- Peptones in digestion, 629.
- „ urine, 789.
- Perforating ulcer of foot in tabes dorsalis, 935.
- „ stomach, 653.
- Perforation of bowel in typhoid fever, 66, 67.
- Periarteritis, 475.
- „ gummatous, 188.
- „ nodosa, 477.
- Pericardial friction, 414.
- Pericarditis, 411.

Pericarditis—

- „ acute, 411.
- „ adhesions, 413.
- „ bacteria, 411, 412.
- „ Bright's disease, 412, 803, 808, 814.
- „ children, 413.
- „ chronic, 416.
- „ adhesions, 416.
- „ angina, 468.
- „ chest retraction, 416.
- „ external form, 416.
- „ heart dilatation or hypertrophy, 416.
- „ dyspnoea, 414.
- „ fever, 414.
- „ fibrinous, 413.
- „ friction, 414.
- „ hæmorrhagic, 413.
- „ myocarditis, 414.
- „ palpitation, 416.
- „ paracentesis, 415.
- „ pneumonia, 412, 510.
- „ primary, 411.
- „ purulent, 413.
- „ rheumatism, acute, 296, 411, 412.
- „ secondary, 411.
- „ syncope, 414.
- „ tubercular, 411, 413, 414.
- „ typhoid and typhus fevers, 415.
- „ ulcer, gastric, 657.

Pericardium, adherent, 416.

Pericardium, diseases, 410.

- „ air, 410.
- „ dropsy, 410.
- „ blood, 410.

Perihepatitis, 768.

Perinephric abscess, 829.

Peripheral neuritis, 1015.

Peristalsis, active, of bowel seen through abdominal wall, 676.

Peritoneum, diseases, 755.

- „ aspiration, 757.
- „ cancer, 769.
- „ circulatory disturbances, 755.
- „ cysts, 770.
- „ dropsy, 4, 755.
- „ inflammation, 757.
- „ new growths, 769.

Peritonitis, acute general, 757.

- „ abdominal muscles, 761, 762.
- „ appendicitis, 689, 758.

Peritonitis—

- „ aged, 761.
- „ bacteria, 759.
- „ Bright's disease, chronic, 759.
- „ children, 761.
- „ collapse, 762, 763.
- „ fever, 763.
- „ gastric ulcer, 656, 657.
- „ hæmatoporphyria, 786.
- „ "hippocratic facies," 760.
- „ intussusception, 674.
- „ leucocytosis, 760, 763.
- „ pain, 761.
- „ pleurisy, 601.
- „ primary, 757.
- „ pulse, 763.
- „ rheumatic, 758.
- „ rigors, 763.
- „ salpingitis, 758, 767.
- „ secondary, 758.
- „ subphrenic abscess, 767.
- „ tenderness, 761.
- „ vomiting, faecal, 762.
- „ wounds, 758.

Peritonitis, chronic, 768.

- „ general, 768.
- „ omentum, 768.
- „ pelvic, 768.
- „ perihepatitis, 768.
- „ perisplenitis, 768.

Perirectal inflammations, 680.

Perityphlitis, 687.

Pernicious anæmia, 352.

- „ anæmia, 354, 356.
- „ ankylostoma, 265, 353.
- „ asthenia, 357.
- „ bone marrow, 356.
- „ cardiac murmurs, 356.
- „ colour index, 354.
- „ cord sclerosis, 356, 864, 943.
- „ dyspnoea, 356.
- „ fever, 356.
- „ hæmogenesis, defective, 354.
- „ hæmolysis, excessive, 354.
- „ hæmorrhages, 356.
- „ iron, 356, 357.
- „ leucopenia, 355.
- „ lymphocytosis, 355.
- „ megalocytes, 354.
- „ megaloblasts, 355.
- „ myelocytes, 355.
- „ normoblasts, 354.
- „ œdema, 356.
- „ primary, 353.
- „ poikilocytes, 354.
- „ polychromatophilia, 355.

- Pernicious anæmia—
 " secondary, 353.
 " uric acid excretion, 783.
 " urobilinuria, 786.
 Pernicious malaria, 242.
 Peroneal type of muscular atrophy, 920.
 Pertussis, 178 (see whooping-cough).
 Pestis, major, 216.
 " minor, 216.
 Petit mal, 973.
 Peyer's patches, in typhoid fever, 59.
 " tuberculosis of intestine, 694.
 Phagocytic action of tonsils, 616.
 Phantom tumor in hysteria, 988.
 Pharyngitis, acute, 617.
 " catarrhal, 617.
 " granular, 618.
 " membranous, 619.
 " phlegmonous, 617.
 " sicca, 618.
 Pharynx, diseases of, 616.
 " angina, 617.
 " inflammations, 617.
 " ulceration, 619.
 Phenylhydrazin test for sugar, 331.
 Phlebitis, 487.
 " suppurative of portal vein, 690, 743.
 Phleboliths, 33.
 Phlebosclerosis, 487.
 Phobias in neurasthenia, 992.
 Phosphates, 785.
 " alkaline, 785.
 " earthy, 785.
 Phosphatic calculi, 796.
 " diabetes, 785.
 Phosphaturia, 784.
 " spermatorrhœa, 785.
 Phosphorus poisoning, 287.
 " anæmia, 288.
 " anuria, 780.
 " cardiac failure, 287.
 " coma, 287.
 " drowsiness, 287.
 " insomnia, 287.
 " jaundice, 287, 741.
 " leucin and tyrosin in urine, 787.
 " resemblance to acute yellow atrophy, 724.
 Phossy jaw in chronic phosphorus poisoning, 288.
 Phrenic nerve paralysis, 1020.
 Phtheiriases, 273.
 Phthisis, chronic, 575.
 " anæmia, 579.
 " aneurismal rupture, 573, 578.
 " bacteria, pyogenic, 574.
 " bronchiectasis, 573.
 " bronchophony, 579.
 " caseation, 571.
 " cavities, 573.
 " signs, 580.
 " cough, 577, 588.
 " cracked pot sound, 580.
 " crepitations, 579.
 " cure, 572, 581.
 " duration of life, 581.
 " dyspnœa, 578, 579.
 " emaciation, 578.
 " fever, 578.
 " fibrosis, 571, 575.
 " fibroid form, 575.
 " gastro-intestinal disturbances, 576, 578.
 " hæmoptysis, 573, 577, 588.
 " hæmatemesis, 577.
 " intestine, 575, 694.
 " " waxy degeneration, 576.
 " larynx, 500, 575.
 " night sweats, 579, 589.
 " opsonic index, 587.
 " pectoriloquy, 580.
 " pleurisy, 574.
 " pneumothorax, 574, 589.
 " quiescence, 572, 574, 581.
 " sputum, 577.
 " " disinfection, 582.
 " toxæmia, 576.
 " treatment of, by cod-liver oil, 585.
 " " open air, 122, 583.
 " tuberculin, 580, 587.
 " vomiting, 578, 579.
 Phthisis, acute, 564.
 " fibroid, 575.
 " florida, 564.
 " pneumonic, acute, 564.
 " renal, 818.
 Physiological albuminuria, 787.
 Pia arachnoid, gummata, 857.
 " inflammation, 844.
 " tumors, 856.
 Pigeon breast, in hypertrophy of tonsils, 623.
 " rickets, 316.
 Pigmentation, 33.
 " autochthonous, 35.
 " bile, 34.

Pigmentation—

- „ blood, 34.
- „ dust, 35.
- „ melanin, 35.
- „ silver, 35.

Pigmentation of skin, in Addison's disease, 405.

- „ arsenic, 1017.
- „ diabetes mellitus, 399.
- „ Hodgkin's disease, 378.
- „ chronic nephritis, 808.
- „ vagabonds, 405.

Pituitary body in acromegaly, 1001.

Pituitous catarrh, chronic, 508.

Plague, 214.

- „ apathy, 217.
- „ bacillus, 214.
- „ „ faeces, sputum and urine, 216.
- „ bubonic form, 216, 217.
- „ bubo, primary, 215, 217.
- „ constipation, 217, 218.
- „ delirium, 217, 218.
- „ epidemic, 215.
- „ fever, 217.
- „ hæmorrhage, perilymphatic, 215.
- „ „ perinephric, 216.
- „ Haffkine's vaccine, 218.
- „ hyperpyrexia, 217, 218.
- „ incubation period, 216.
- „ infection, methods, 214.
- „ lymphatic glands, 215.
- „ major, 216.
- „ minor, 216.
- „ pneumonic, 216.
- „ rats, 214, 215, 218.
- „ septicæmic, 216.
- „ skin necrosis, 217.
- „ spleen enlargement, 216.
- „ Yersin's serum, 218.

Plasmodium malaria, 237.

Plastic bronchitis, 509.

- „ bronchial casts, 510.
- „ dyspnœa, 510.

Pleura, diseases of, 594.

- „ air, 596.
- „ blood, 595.
- „ chyle, 596.
- „ dropsy, 594.
- „ empyema, 605.
- „ inflammations, 598.
- „ parasites, 607.
- „ tumors, 606.

Pleural effusions, 594, 595, 596, 600.

- „ bacteriology, 600, 601.
- „ cytology, 600.

Pleural effusions—

- „ pulmonary collapse, 602.
- „ paracentesis, 604.

Pleurisy, 598.

- „ ægophony, 603.
- „ bacteria, 599.
- „ breathing, rapid and shallow, 602.
- „ Bright's disease, 598, 814.
- „ cardiac failure, 603.
- „ chronic, 606.
- „ dry, 600.
- „ effusions pleural, cytological characters, 600.
- „ collapse of lung, 602.
- „ fever, 602.
- „ fibrinous, 600.
- „ friction, 603.
- „ hæmorrhagic, 601.
- „ influenza, 599.
- „ pain in side, 602.
- „ paracentesis, 604.
- „ perinephric abscess, 829.
- „ phthisis, 565, 566, 574.
- „ posture of patient, 602, 603.
- „ pneumonia, 540, 599.
- „ primary, 599.
- „ purulent, 601.
- „ rheumatic fever, 296.
- „ secondary, 598.
- „ serous, 600.
- „ subphrenic abscess, 766.
- „ tubercular, 599.
- „ typhoid, 66.
- „ gastric ulcer, 657.

Pleuritis, 598.

Pleurodynia, 603.

Pleuropneumonia, 234.

Plexiform neuroma, 1019.

Plumbism, 289.

Pneumococcus, 535.

Pneumogastric nerve, 1059.

- „ branches, cardiac, 1061.
- „ „ gastric, 1062.
- „ „ laryngeal, 1060.
- „ „ œsophageal, 1062.
- „ „ palatal, 1060.
- „ „ pharyngeal, 1059.
- „ „ pulmonary, 1062.

Pneumonia, actinomycotic, 131, 591.

- „ albuminuria, 537, 538, 788.
- „ albumosuria, 789.
- „ arthritis, 540.
- „ asphyxia, 541.
- „ aspiration, 547, 549.
- „ bacteria, 535, 546, 549, 558, 559, 562.

Pneumonia—

- „ breathing, rapid and shallow, 538.
- „ Bright's disease, chronic, 814.
- „ bronchitis, 505.
- „ bronchogenic, 533.
- „ bronchophony, 539.
- „ cancrum oris, 613.
- „ caseous acute, 564.
- „ catarrhal, 543.
- „ children, 539.
- „ chlorides, excretion, 537, 538, 785.
- „ chronic, 554.
- „ coma, 539.
- „ convulsions, 977.
- „ cough, 538, 539.
- „ crepitations, 539.
- „ crisis, 538.
- „ croupous, 534.
- „ cyanosis, 541.
- „ delirium, 539.
- „ diabetes, 330, 552.
- „ drunkards, 539, 541.
- „ dullness percussion, 539.
- „ empyema, 540.
- „ endocarditis, 540.
- „ fever, 537, 539.
- „ fibrinous, 534.
- „ Frankel's diplococcus, 535.
- „ gangrene of lung, 552.
- „ grave signs, 541.
- „ hematogenic, 533.
- „ heart, 537, 541.
- „ hepatisation, 535.
- „ herpes of lips, 538.
- „ hyperpyrexia, 541.
- „ hypostatic, 547, 549.
- „ influenzal, 90, 545, 559.
- „ interstitial, 554, 562, 571.
- „ jaundice, 540.
- „ kidneys, 537, 540, 800.
- „ leucocytosis, 537, 541.
- „ lobar, 534.
- „ lobular, 543.
- „ massive form, 539.
- „ meningitis, 539, 540, 853.
- „ neuritis, peripheral, 540.
- „ old people, 539.
- „ parotitis, 540.
- „ pericarditis, 412, 540.
- „ peritonitis, 759.
- „ pleurisy, 540, 599.
- „ pleurogenic, 533.
- „ prostration, 538.
- „ pseudo lobar, 544.

Pneumonia—

- „ pulse respiration, ratio, 538.
- „ purulent, 549.
- „ rigors, 537.
- „ spleen, 537.
- „ sputum, absent, 539.
- „ „ dark liquid, 541.
- „ „ rusty, 538.
- „ strabismus, 539.
- „ syphilitic, 590.
- „ thrombosis, intracardiac, 537, 542.
- „ typhoid, 61, 65, 546, 558.
- „ tubercular, 564, 570.
- „ urea increased, 538.
- „ varieties, 546.
- „ vomiting, 539.
- „ white, 590.
- Pneumonitis, 535.
- Pneumonoconiosis, 555.
- Pneumopericardium, 410.
- Pneumothorax, 596.
- „ bacillus lactis aerogenes, 596.
- „ empyema, 601.
- „ gas, 596.
- „ paracentesis, 605.
- „ phthisis, chronic, 574, 583.
- „ splashing sound, 597.
- Poikilocytes, 342.
- Poisoning by alcohol, 274.
- „ arsenic, 288.
- „ cocaine, 285.
- „ food, 279.
- „ lead, 289.
- „ mushrooms, 280.
- „ opium, 283.
- „ phosphorus, 287.
- „ ptomaines, 279, 385.
- „ snakes, 281.
- „ tobacco, 285.
- Polychromatophulia, 342, 355.
- Poliomyelitis, acute, 904.
- „ adults, 910.
- „ age, 904, 910.
- „ anterior horn cells, 906.
- „ baby limbs, 908.
- „ convulsions, 907.
- „ degeneration, secondary, 906.
- „ deformity, 908.
- „ fever, 907.
- „ galvanism, 909.
- „ leptomeningitis, 905.
- „ lesion extent, 905, 906.
- „ motor paralysis, 907.
- „ onset, 907.

- Poliomyelitis, acute—
 " pain, 907.
 " reaction of degeneration, 908.
 " reflexes, 908.
 " sclerosis, 907.
 " sex, 904.
 Poliomyelitis, anterior chronic, 913.
 Poliomyelitis, subacute, in adults, 910.
 Polyuria, in amyloid degeneration of kidney, 755.
 " diabetes, 330, 338, 779.
 " granular contracted kidney, 779, 815.
 Pons, lesions, 838, 874, 955.
 Popliteal nerves, lesions, 1027.
 Porencephaly, 887, 924.
 Portal vein, circulation retarded, 720.
 " suppurative phlebitis, 690, 743.
 Post-hemiplegic, athetotic movements, 875.
 " chorea, 875.
 Posterior spinal sclerosis, 929 (*see* tabes dorsalis).
 Pott's disease, 897.
 Poynton and Paines diplococcus rheumaticus, 294.
 Pregnancy, and Bright's disease, 800.
 " chorea, 967.
 " relapsing fever, 54.
 " small-pox, 152, 155.
 " syphilis, 189.
 " yellow atrophy, 727.
 Presystolic murmur, 450.
 " thrill, 450.
 Priapism in leukæmia, 368.
 Proctitis, 679, 680.
 Professional spasms, 978.
 Proglottides of tape worm, 256.
 Progressive muscular atrophy, 913.
 " claw-like hand, 916.
 " lordosis, 916.
 " onset, 914.
 " paralysis, 916.
 " spasticity, 916.
 " "main en griffe," 916.
 Progressive neural atrophy, 920.
 " club-foot, 920.
 Prophylaxis against cholera, 85.
 " malaria, 245.
 " plague, 218.
 " scurvy, 385.
 " tuberculosis, 121.
 " yellow fever, 205.
 Proptosis in exophthalmic goitre, 397, 398.
 Protozoa, diseases due to, 236.
 Prune-juice expectoration in lung gangrene, 553.
 Pruritus, diabetes mellitus, 335.
 Psammoma, 857.
 Pseudo bulbar paralysis, 919, 960.
 " diarrhœa, 675.
 " hydrophobia, 230.
 " hypertrophic paralysis.
 " lobar pneumonia, 544.
 " leukæmia, 370.
 " ptosis, 1038.
 " tubercle caused by coccidia, 249.
 Pseudo-hypertrophic paralysis, 32, 921.
 " age, 921.
 " heredity, 921.
 " posture, 922.
 " sex, 921.
 Psilosis, 684.
 Psoas abscess, 119, 900.
 Psorospermiasis, 248.
 " cutaneous, 249.
 " internal, 249.
 Psychical disturbances in acute chorea, 967.
 " hysteria, 989.
 " neurasthenia, 992.
 Ptomaine poisoning, 279.
 Ptosis, 1037.
 " causes, 1037.
 " cerebral origin, 1038.
 " hysterical, 1038.
 " morning, in women, 1038.
 " progressive muscular atrophy, 1038.
 " pseudo, 1038.
 Pytalism, 615.
 Puerperal fever, 143.
 " lung abscess, 549.
 Pulmonary apoplexy, 530.
 " artery aneurisms, 577.
 " gymnastics, 517.
 " hæmorrhage, 528.
 " incompetence, 457.
 " osteo - arthropathy hypertrophic, 1002.
 " stenosis, 457.
 " tuberculosis, 561.
 Pulmonary diseases, 518 (*see* lung diseases of).
 Pulse, 471.
 " aortic disease, 453, 455.
 " bigeminus, 463.

Pulse—

- capillary, 453.
- Corrigan's, 453.
- delayed, 472.
- dicotism, 472.
- typhoid fever, 61.
- intermittent, 472.
- irregularities, 472.
- mitral stenosis, 450.
- radial aneurism, 484.
- rate, in adults, children and
foetus, 471.
- acute bronchitis in
very young and
old people, 505.
- respiration ratio in pneu-
monia, 538.
- wave, anacrotic, 473.
- dicrotic, 452.
- predicrotic, 452.
- water-hammer, 453.

Pupil, Argyll Robertson, 1038.

- brain tumor, 956.
- general paralysis, 947, 1038.
- tabes dorsalis, 934, 1038.
- dilatation, 1037.
- pin point, 934, 955.
- Wernicke's reaction, 1036.

Purpura, 381.

- arthritic, 382.
- asphyxia, 382.
- fulminans, 383.
- hæmorrhagica, 383.
- Henoch's, 383.
- infections, 381, 382.
- neurotic, 382.
- peliosis rheumatica, 382.
- pemphigoid, 383.
- rheumatica, 382.
- simplex, 382.
- stigmata, 382.
- symptomatic, 381.

Purulent pneumonia, 549.

Pus in urine, 790.

Pustule, malignant, 222.

Putrid sore mouth, 611.

Pyæmia, 136.

- abscesses, 136, 137, 138, 549.
- arthritis, 138.
- blood spread, 137.
- cellulitis, 137.
- emboli, 136.
- endocarditis, 440.
- erysipelas, 141.
- hepatitis suppurative, 710,
712, 713.
- lymphangitis, 137.

Pyæmia—

- meningitis, 138.
- nephritis, 800, 806.
- pericarditis, 138.
- peritonitis, 759.
- phlebitis, 136.
- pyelophlebitis, 137.
- skin eruptions, 138.
- sweating, 138.
- thrombosis, 136.
- typhoid state, 138.

Pyæmic abscess of liver, 713.

Pyelitis, 819.

- abscesses, renal, 821.
- perinephric, 829.
- albuminuria, 821.
- anæmias, 820.
- bacteria, 820.
- Bilharzia ova, 255, 820.
- calculus, 820.
- hæmaturia, 821.

Pyelonephritis, 820.

Pylephlebitis suppurativa in actin-
omycosis of liver, 130, 711.

- amœbic dysentery, 711.
- appendicitis, 690.
- emboli, suppurative of portal
vein, 709.
- pyæmia, 137.

Pylorectomy, 652, 667.

Pyloric tumors, 666.

Pyogenic cocci, endocarditis, 433.

- hepatic abscess, 711.
- leptomeningitis secondary,
855.
- nasal catarrh, 489.
- pericarditis, 411.
- peritonitis, 759.

Pyonephrosis, 821.

Pyo-pneumothorax subphrenicus,
656, 766.

Pyorrhœa alveolaris, 611.

Pyramidal tract, 835.

Pyramids of medulla, 835.

Pyrexia, 43 (*see* fever and hyper-
pyrexia).

Pyrogenetic substance, 46.

Pyrosis, 632.

Pyuria, 788, 790.

- cystitis, 790.
- perinephric abscess, 830.
- pyelitis, 790, 821.
- renal tuberculosis, 790, 819.

Quarantine in cholera, 85.

- plague, 218.
- yellow fever, 205.

Quartan ague, 238.
 Quincke's hydrocephalus, 858.
 Quinine in malaria 245.
 Quinsy, 620.
 " abscess, 621.
 " cervical glands, 621.
 " delirium, 621.
 " fever, 621.
 " gangrene of pharynx, 621.
 " hæmorrhage, 621.
 " nasal regurgitation, 621.
 " nephritis, 800.
 " suffocation, 621.
 Quotidian ague, from double ter-
 tian parasites, 239.
 " triple quartan parasites, 239.
 " malignant, 238.
 Rabies, 225.
 " animals, 225.
 " asphyxia, 229.
 " delirium, 228.
 " dumb, 228.
 " fever, 229.
 " furious, 227.
 " geographical distribution,
 226.
 " hyperæsthesia, 229.
 " incubation period, 227.
 " infection methods, 226.
 " Landry's paralysis resem-
 blance, 229.
 " man, 228.
 " mania, 229.
 " Negri bodies, 229.
 " opisthotonos, 229.
 " paralytic, 228.
 " Pasteur's treatment, 231.
 " speech, 228.
 " swallowing difficulty, 228,
 229.
 " thirst, 228.
 " virus, 226.
 " virus fixé, 232.
 Railway brain, 994.
 " spine, 994.
 Rapid heart, 464.
 Ranula, 615.
 Rash, chicken pox, 162.
 " chloral hydrate and copaiba,
 176.
 " erysipelas, 140.
 " German measles, 176.
 " glanders, 220.
 " measles, 173.
 " mulberry coloured in typhus
 fever, 195.

Rash—
 " pyæmia, 138.
 " relapsing fever (rare), 53.
 " scarlet fever, 165.
 " small-pox, 151.
 " syphilis, 186, 188.
 " table of, in eruptive fevers,
 147.
 " typhoid fever, 64.
 " vaccination, 161.
 Ray fungus, 129 (*see* actinomycosis).
 Raynaud's disease, 996.
 " asphyxia, local, 997.
 " chilblains, 997.
 " cold, 998.
 " erythromelalgia, 999.
 " fatigue, 995.
 " gangrene, local, 997.
 " multiple, 997.
 " hæmoglobinuria, paroxys-
 mal, 388, 998.
 " hemiplegia, transient, 998.
 " nephrorraphy, 998.
 " scleroderma, 998, 1000.
 " spasm of arteries and arteri-
 oles, 996.
 " syncope, local, 997.
 " symmetry of lesions, 996.
 Reaction of degeneration, 839.
 Recklinghausen's disease, 1019.
 Rectal crises in tabes dorsalis, 935.
 Rectum, inflammation, 680.
 " syphilitic disease, 697.
 " tubercular disease 697.
 Rectum, inflammation, 680.
 " " fistula, 680, 697.
 " " ischio-rectal abscess,
 680.
 " " mucous discharges,
 680.
 " " tenesmus, 680.
 Recurrent laryngeal nerve paralysis,
 1061.
 Recurring utterances, 970.
 Reflex action, false, 841.
 " true, 839.
 Reflex arc, 841, 842.
 " injury, 841.
 Reflexes deep, 840.
 " ankle clonus, 840.
 " knee jerk, 840.
 " triceps, 840.
 " superficial, 839.
 " " abdominal, 840.
 " " Babin'ski's, 875.
 " " conjunctival, 840.
 " " cremasteric, 840.

Reflexes, superficial—

- „ „ gluteal, 840.
- „ „ plantar, 840.
- „ „ pupil, 840.
- „ „ scapular, 840.

Reflexes, ataxic paraplegia, 940

- „ cerebral hæmorrhage, 875.
- „ cerebellar ataxia, 943.
- „ Friedreich's ataxia, 942.
- „ general paralysis, 947.
- „ Landry's paralysis, 911.
- „ myelitis, 900.
- „ peripheral neuritis, alcoholic, 1017.
- „ poliomyelitis, acute, 908.
- „ progressive muscular atrophy, 916.
- „ primary spastic paraplegia, 926.
- „ spinal hæmorrhage, 880.
- „ syringomyelia, 863.
- „ tabes dorsalis, 945.

Reflexes deep, exaggerated, in cerebral hæmorrhage, 875.

- „ cerebellar ataxia, 943.
- „ myelitis, 900.
- „ primary spastic paraplegia, 926.
- „ syringomyelia, 863.

Reflexes deep, lost, in general paralysis (sometimes), 947.

- „ Landry's paralysis, 911.
- „ peripheral neuritis, alcoholic, 1017.
- „ acute poliomyelitis, 908.
- „ progressive muscular atrophy (gradually), 916.
- „ tabes dorsalis, 945.

Regurgitation of blood in heart, 444.

Relapse in typhoid fever, 64.

Relapsing fever, 52.

- „ abortion, 54.
- „ cough, 53.
- „ delirium, 54.
- „ epistaxis, 54.
- „ fever, 53.
- „ geographical distribution, 55.
- „ hæmaturia, 54.
- „ jaundice, 53.
- „ immunity, 52.
- „ liver enlargement, 53.
- „ nephritis, 54.
- „ peritonitis, 54.
- „ period of incubation, 53.
- „ pneumonia, 54.
- „ rash, 53.
- „ relapses, 54.

Relapsing fever—

- „ spirillum, 52.
- „ spleen, 52, 53.

Remittent fever, 46.

- „ malarial, 244.

Renal calculi, 795.

Renal colic, 735, 797.

- „ gravel, 797.

Resonance, vocal, in chronic phthisis, 579.

- „ pneumothorax, 557.

Respiratory system, diseases, 488.

Retina affections, 1034.

Retinitis, albuminuric, 804, 1034.

- „ leukæmic, 368.

- „ syphilitic, 186.

Retraction of head in meningitis, tubercular, 852.

Retro-pharyngeal abscess, 617.

Retropulsion in paralysis agitans, 964.

Retzius's anastomosis, 718.

Rhabdomyoma, rhabdomyosarcoma of kidney, 826

Rheumatic fever, rheumatism acute, 293.

- „ anæmia, 300.
- „ arthritis, 295.
- „ blood coagulability increased, 295.
- „ cardiac dilatation, 297.
- „ „ failure, 298.
- „ children, 297, 298.
- „ chill, 293.
- „ chorea, 297.
- „ diplococcus, 294.
- „ endocarditis, 296, 297.
- „ fever, 295.
- „ heredity, 293.
- „ hæmatoporphyria, 786.
- „ hyperpyrexia, 296, 300.
- „ iodine, 301.
- „ iritis, 296.
- „ leucocytosis, 295.
- „ meningitis, 852.
- „ metastasis, 296, 298.
- „ micro-organisms, 294.
- „ myocarditis, 297.
- „ nephritis, 800.
- „ pericarditis, 296, 297.
- „ perspiration, 296.
- „ pleurisy, 296.
- „ relapse, 297.
- „ salicylates, 299.
- „ subcutaneous nodules, 297.
- „ sudamina, 296.
- „ tonsillitis, 294.

- Rheumatic gout, 307 (*see* arthritis deformans).
- Rheumatism, chronic articular, 301.
- „ muscular, 303.
- Rheumatoid arthritis 307 (*see* arthritis deformans).
- Rhinitis, acute, 488.
- Resection of ribs in empyema, 605.
- Rice water stools in cholera, 83.
- Rickets, 313.
- „ age, 313.
- „ anæmia, 316.
- „ belly, big, 316.
- „ bow legs, 316.
- „ bronchitis, 316.
- „ collapse pulmonary, 524.
- „ convulsions, 317, 977.
- „ craniotabes, 315.
- „ diarrhœa, 317.
- „ diet, 313.
- „ epiphysis, 314.
- „ fontanelles, 315.
- „ knock knee, 316.
- „ laryngismus stridulus, 316, 317, 502.
- „ liver, 316.
- „ long bones, 315, 316.
- „ meat, raw, 317.
- „ nystagmus, 317.
- „ pelvis, 315.
- „ periosteum, 314.
- „ perspiration of head, 316.
- „ pigeon breast, 316.
- „ rosary, 315, 316.
- „ spleen, 316.
- „ tetany, 317, 980, 982.
- „ tissues affected, 314.
- Rigidity, early, 875.
- „ late, 875.
- Rigors, malaria, 243.
- „ pneumonia, 537.
- „ scarlet fever, 164.
- „ small-pox, 150.
- „ typhoid fever, 61.
- „ tubercular meningitis, 852.
- Risus sardonius, 107.
- Rock fever, 87.
- Romberg's sign, in ataxic paraplegia, 940.
- „ Friedreich's ataxia, 942.
- „ tabes dorsalis, 933.
- Roots of nerves, pressure on, in meningitis, tubercular, 852.
- Rosary, rickety, 315, 316.
- Rose, 139 (*see* erysipelas).
- Rose-coloured rash of typhoid fever, 64.
- Roseola, 175 (*see* German measles).
- Rotheln, 175 (*see* German measles).
- Round worms, 261.
- Rubella, 175 (*see* German measles).
- Rubeola, 172 (*see* measles).
- Rupture of heart, 419, 424.
- Rusty sputum in pneumonia, 538.
- Rye-bread poisoning, 279.
- Saccharomyces albicans, 610.
- Sach's amaurotic family idiocy, 927.
- Sacral plexus, lesions, 1027.
- St Anthony's fire, 139.
- St Vitus's dance, 965, 969.
- Salaam, chorea, 969.
- „ movements in hysteria, 988.
- Saliva, secretion of, diminished, 615.
- „ „ increased, 615.
- „ bulbar paralysis, 919.
- Salivary glands, diseases of, 615.
- „ inflammation, 615.
- „ tumors, 615.
- Saltatory spasm, 969.
- Sanatoria for consumptives, 582.
- Sappey's accessory portal system of veins, 717.
- Sapromia, 133.
- „ abscesses, 134.
- „ collapse, 134.
- „ diphtheria, 132.
- „ puerperal fever, 143.
- „ tetanus, 134.
- Sarcocystis miescheri, 249.
- Sarcoma, brain, 952.
- „ heart, 431.
- „ kidney, 826.
- „ liver, 728.
- „ lung, 591.
- „ melanotic, 35.
- Sarcoptes scabei, 272.
- Sausage poisoning, 279.
- Scabies, 272.
- Scarlet fever, 163.
- „ albuminuria, 165-168, 788.
- „ age, 164.
- „ anginosa, 164.
- „ cervical glands, 167.
- „ coma, 168.
- „ convulsions, 166, 168, 977.
- „ cows, 163.
- „ deafness, 168, 171.
- „ desquamation, 167.
- „ diarrhœa, 168.
- „ diphtheria, 168.
- „ diplococcus of Class, 163.
- „ dropsy, 168.
- „ epistaxis, 165.

- Scarlet fever—
 „ false membrane, 96, 157.
 „ fever, 165, 166.
 „ hyperpyrexia, 170.
 „ immunity, 169.
 „ incubation period, 164.
 „ infectious modes, 164.
 „ leucocytosis, 169.
 „ malignant, 164.
 „ nephritis, 168, 171, 798, 799.
 800, 802.
 „ otitis media, 168, 171.
 „ pruritus, 167.
 „ rash, characters, 165.
 „ absence, 165.
 „ simplex, 164.
 „ sore throat, 164, 166, 167.
 „ streptococcus of Klein, 163.
 „ tongue, strawberry, 166.
 „ tonsils enlargement, 167.
 „ wet pack, 170.
 Scarlatina rheumatica, 205.
 School made chorea, 965.
 Sciatica, 1010.
 Sciatic nerve lesions, 1027.
 Scleroderma, 999.
 Sclerose en plaques, 948.
 Sclerosis of brain and cord, 913.
 „ amyotrophic, lateral, 914.
 „ anæmic, 943.
 „ cerebro-spinal, multiple, 948.
 „ diabetic, 944.
 „ diffuse, 913.
 „ disseminated, 948.
 „ insular, 948.
 „ posterior spinal, 929.
 „ primary lateral, 926.
 „ toxic, 943.
 Scolex stage of tapeworms, 256.
 Scolices of echinococcus, 259.
 Scoliosis in syringomyelia, 863.
 Scorbutus, 384.
 Scrivener's palsy, 978.
 Scotoma, central, 1033.
 „ peripheral, 1032.
 Scrofulous disease, 115.
 Scurvy, 384.
 „ brawny swellings, 385.
 „ diet, 384.
 „ epistaxis, 385.
 „ gums, 385.
 „ hæmorrhages, 385.
 „ leucocytosis, 385.
 „ night blindness, 385.
 „ syncope, 385.
 Scurvy, infantile, 386.
 „ attitude, 387.
 Scurvy, infantile—
 „ rickets, signs, 386.
 Scybala, 676.
 Seasonal relations, follicular tonsil-
 litis, 620.
 „ hay fever, 492.
 Secondary contracture in hemiplegia,
 875.
 Secondary deviation of normal eye
 in paralysis of other eye,
 1041.
 Secondary fever of small-pox, 150.
 Senile emphysema, 520.
 Sensory system, 841.
 Septicæmia, 135.
 „ anthrax in animals, 136.
 „ blood, 135.
 „ endocarditis, 439.
 „ germs, 135.
 „ nephritis, 800.
 „ pneumococcal infection in
 guinea pigs, 136.
 Serratus paralysis, 1024.
 Serumtherapy (*see* blood serum).
 Seven day fever, 52.
 Sex influence, asthma, 514.
 „ chlorosis, 349.
 „ chorea, 965.
 „ diabetes insipidus, 338.
 „ exophthalmic goitre, 396.
 „ gall stones, 731.
 „ general paralysis, 945.
 „ gout, 318, 326.
 „ hæmophilia, 379.
 „ hysteria, 985.
 „ liver, yellow atrophy, 724.
 „ myxœdema, 395.
 „ poliomyelitis, acute, 904.
 Shaking palsy, 962.
 Shingles, 938.
 Ship fever, 192.
 Shock, a cause of traumatic neuroses,
 994.
 Sick headache, 983.
 Sleeping sickness, 250.
 „ cerebro-spinal fluid, 252.
 „ coma, 253.
 „ complications, 253.
 „ diplococcus, 250.
 „ epileptic fits, 253.
 „ emaciation, 253.
 „ eruptions, 253.
 „ fever, 252.
 „ geographical distribution,
 250.
 „ glossina palpalis, 251.
 „ knee jerks, 253.

Sleeping sickness—

- „ lethargy, 253.
- „ lymphocytosis, 252.
- „ lymphatic glands, 252.
- „ lumbar puncture, 253.
- „ meningitis, 252.
- „ tremors, 253.
- „ trypanosoma, 250, 251, 252.

Siderosis, 35, 556.

Signal symptom in cortical lesions, 953.

Silicosis, 556.

Singer's node, 499.

Sinuses, cerebral thrombosis, anæmia and chlorosis, 889.

- „ ear disease, 889.
- „ marantic, 889.
- „ primary, 889.
- „ secondary, 889.

Sixth nerve paralysis, 1040.

Skin itching, in German measles, 176.

- „ jaundice, 739.

Skull, hereditary syphilis, 189.

- „ hydrocephalus, 860.
- „ rickets, 315.
- „ percussion, 895.

Slow heart, 466.

Small-pox, 149.

- „ abortion, 152, 155.
- „ abscesses, cutaneous, 153.
- „ albuminuria, 151, 152.
- „ asthenia, 155.
- „ benign, 149.
- „ blindness, 153.
- „ coma, 153.
- „ convulsions in children, 150, 153.
- „ confluent, 149.
- „ contagion, strength, 155.
- „ corymbous, 149.
- „ deafness, 153.
- „ delirium, 151, 156.
- „ discrete, 149.
- „ eruption, characters, 151.
- „ „ absence, 149.
- „ eye affections, 153, 156.
- „ fever, primary, 150.
- „ „ secondary, 150, 152.
- „ hæmorrhage, 149.
- „ inoculation, 149.
- „ light, 156.
- „ leucocytosis, 152.
- „ malignant, 149.
- „ mania in adults, 153.
- „ myocarditis, 152.
- „ nephritis, 800.

Small-pox—

- „ oophoritis, 152.
- „ orchitis, 152.
- „ pains in back, 150, 151.
- „ parotitis, 151.
- „ pneumonia, lobular, 152.
- „ rashes in initial stage, measles-like and scarlet fever-like, 152.
- „ skin treatment, 156, 157.
- „ smell, bodily, 151.
- „ stages, 150.
- „ sweating, 150.
- „ tonsillitis, 151.
- „ urine, 151.
- „ vaccination, 149, 155, 159, 161.

Snake poisoning, coluber group, 281.

- „ paralysis of respiration, 282.

Snake poisoning, viperine group, albuminuria, 282.

- „ cardiac depression, 282.
- „ hæmolysis, 282.
- „ antivenom of Calmette and Fraser, 282.

Snuffles, 189.

Soil, ground water of and cholera, 81.

Solvent treatment of renal calculi by drinking large quantities of water, 797.

Sordes in mouth, 64.

Sore throat, 617.

- „ German measles, 176.
- „ scarlet fever, 164.

Spasmodic laryngitis, 502.

- „ wryneck, 1065.

Spasms, chorea, 966.

- „ epilepsy, 972.
- „ clonic and tonic in epilepsy, 972.
- „ epidemic cerebro-spinal meningitis, 78.
- „ ergotism, 279.
- „ hydrophobia, 228, 233.
- „ hysteria, 986.
- „ Jacksonian, in lesions of facial nerve, 1052.
- „ nystagmus, 1042.
- „ professional, 978.
- „ saltatory, 969.
- „ tetanus, 106, 233.

Spastic diplegia in infants, 924.

Spastic paraplegia, in adults, 926, 927.

- „ syphilitic spinal, 927.
- „ children, 925.

- Spastic paraplegia—
 „ hysteric, 927.
 „ primary, 926.
- Special symptoms, treatment of in
 typhoid fever, 71.
- Specific nephritis, 818.
 „ pneumonia, 534.
- Speech, disorders of, 957 (*see*
 aphasia).
- Speech, in adenoids, 495, 623.
 „ bulbar paralysis, 918.
 „ cerebral hæmorrhage, 960.
 „ delirium tremens, 276.
 „ disseminated sclerosis, 950.
 „ general paralysis, 946.
 „ hydrophobia, 228.
 „ myxœdema, 394.
 „ paralysis agitans, 964.
 „ scanning, 950.
- Sphygmograph, 472.
 Sphygmometer, 473.
- Spina bifida, 865.
 „ sac contents, 865.
 „ talipes, 865.
- Spinal accessory nerve lesions, 1063.
- Spinal concussion, 994.
- Spinal cord, anæmia, 866.
 „ atrophy, 683.
 „ compression, 880, 897.
 „ congestion, 865.
 „ degenerations, 837.
 „ embolism and thrombosis of
 vessels, 886.
 „ endarteritis, 886.
 „ fissures, 883.
 „ hæmorrhage, into mem-
 branes, 879.
 „ „ substance, 881.
 „ inflammations, membranes,
 845, 849.
 „ „ substance (myelitis).
 897.
 „ lepto-meningitis, acute, 849.
 „ „ chronic, 856.
 „ „ tubercular, 850.
 „ pachymeningitis externa,
 845.
 „ „ interna, 847.
 „ sclerosis, 913.
 „ „ anæmic, 943.
 „ „ lateral, 926.
 „ „ insular, 948.
 „ section, complete, 843.
 „ „ hemi-, 843.
 „ tumors, dural, 848.
 „ „ pial, 856.
 „ „ substance, 951.
- Spinal membranes, hæmorrhage, 879.
 „ inflammations, 845-849.
 „ tumors, 848, 856.
- Spinal nerves, diseases of, 1020.
- Spinal paralysis, atrophic, 904.
- Spiral's, Curschmann's, 515.
- Spirillum fever, 52.
- Spirochæte of Obermeyer, 52.
- Spleen, alcoholism, 275.
 „ anthrax, 222.
 „ Banti's disease, 371.
 „ cancer, 864.
 „ chloroma, 373.
 „ degeneration, waxy, 29.
 „ diphtheria, 29.
 „ embolism, 12, 139.
 „ excision, in splenic anæmia
 of adults, 371.
 „ exophthalmic goitre, 397.
 „ hepatic cirrhosis, 718, 720,
 721.
 „ infarct, 12.
 „ leprosy, 124.
 „ leukæmia, 365, 366, 368.
 „ lymphadenoma, 376.
 „ malaria, 242, 245.
 „ Malta fever, 87, 88.
 „ enlargement in plague, 216.
 „ relapsing fever, 52, 53.
 „ rheumatic arthritis, chronic,
 310.
 „ rickets, 316.
 „ splenic anæmia, children,
 370.
 „ „ adults, 371.
 „ syphilis, 189.
 „ typhoid fever, 60, 63.
 „ tuberculosis, acute miliary,
 562, 564.
 „ „ acute caseous, 569.
 „ „ chronic caseous, 576.
 „ venous congestion, in aortic
 incompetence, 411.
- Splenic anæmia, 370.
 „ fever, 222.
- Spitting of lips in apoplexy, 172.
- Spotted fever, 77.
- Sprue, 684, 685.
 „ asthenia, 605.
 „ diet, 685.
 „ liver, 684, 685.
 „ stomatitis, aphthous, 614.
 „ stools, 694, 695.
- Sputum, actinomyces, 511.
 „ albuminous, 605.
 „ amaranth, 712.
 „ bronchial cancer, 510.

Sputum—

- „ Charcot's crystals, 515.
- „ coctum, 504.
- „ crudum, 504.
- „ Curschmann's spirals, 515.
- „ disinfection, in tuberculosis, 582.
- „ Dittrich's plugs, 513.
- „ elastic fibres, 577.
- „ foetid, 553.
- „ gangrene of lung, 553.
- „ hæmorrhagic, 448, 531.
- „ hydatid cysts or hooklets, 593.
- „ influenza bacillus, 90.
- „ nummular, 508, 577.
- „ plague bacillus, 216, 217.
- „ pneumococcus, 536.
- „ prune-juice coloured, 553, 592.
- „ purulent, 550.
- „ rusty, 538.
- „ Traube's plugs, 513.
- „ tubercular bacillus, 563, 569, 577.
- „ significance, 120; of numbers, 577.

Squint in hypermetropia, 1042.

Staphylococci, in appendicitis, 688.

- „ brain abscess, 893.
- „ bronchitis, acute, 503.
- „ diphtheria, 94.
- „ endocarditis, 433.
- „ peritonitis, 759.
- „ pleurisy, 599.
- „ pneumonia, 533, 546.

Status epilepticus, in epilepsy, 973.

„ uræmia, 783.

Stellwag's sign in exophthalmic goitre, 398.

Stenosis, of aorta, 409.

- „ aortic valves, 426, 454.
- „ bile ducts, 745.
- „ coronary arteries, 417, 419, 423, 424, 467.
- „ larynx, 500.
- „ mitral valves, 449.
- „ œsophagus, 625.
- „ pulmonary artery and valves, 409, 426, 457.
- „ trachea and bronchi, 517.
- „ tricuspid valves, 426, 456.

Steppage, gait, 1016, 1027.

Stercoraceous vomiting, 676.

Stercoral ulcers, 684.

Stitch in side in pleurisy, 602.

Stomach, cancer of, 661.

Stomach, cancer of—

- „ age, 651.
- „ anæmia, 664, 665.
- „ appetite, 664.
- „ asthenia, 667.
- „ cachexia, 666.
- „ emaciation, 664.
- „ fever, 665.
- „ hæmatemesis, 665, 667.
- „ hiccough, 667.
- „ hydrochloric acid, 665.
- „ liver, 664, 666.
- „ lymphatic glands, 664, 666.
- „ metastases, 664.
- „ œdema, 665.
- „ pain, 665, 667.
- „ subcutaneous nodules, 664.
- „ tumor, 666.
- „ ulcer, 663.
- „ urea, diminution, 781.
- „ vomiting, 664.

Stomach, dilatation of, acute, 650.

- „ „ death, 650.
- „ „ dyspnoea, 650.
- „ „ cardiac embarrassment, 650.
- „ „ chronic, 650.
- „ „ atony of stomach wall, 651, 652.
- „ „ catarrh, 644, 651.
- „ distension, epigastric, 651.
- „ flatulence, 651.
- „ hyperacidity, 651.
- „ kidney, movable, 722.
- „ lavage, 652.
- „ oxaluria, 795.
- „ peristalsis, 652.
- „ splashings after food, 652.
- „ tetany, 651, 980, 981.
- „ vomit with bacteria and sarcina, 651.

Stomach, diseases, cancer, 661.

- „ degenerations, 636.
- „ dilatation, 650.
- „ displacement, 637.
- „ hour-glass, 637, 656.
- „ inflammation, 638-650.
- „ leather bottle, 643.
- „ stenosis of pylorus, 637.
- „ ulcer, 653.

Stomach contents, examination, 646.

Stomatitis, 608.

- „ actinomycotic, 130, 614.
- „ aphthous, 609.
- „ catarrhal, 608.
- „ foetid, 611, 612.

Stomatitis—

- „ gangrenous, 612.
- „ „ bacteria, 612.
- „ measles, 613.
- „ parasitic, 610.
- „ oidium or saccharomyces albicans, 610.
- „ suppurative, 613.
- „ „ Bright's disease, 613.
- „ syphilitic, 614.
- „ tubercular, 614.
- „ ulcerative, 610.

Stools, of cholera, rice water, 83.

- „ dysentery, "meat washings," "boiled eggs" or "frog's spawn," 209.
- „ bile duct obstruction, clay-coloured, 741.
- „ chronic gastric catarrh, clay-coloured, 641.
- „ intestinal hæmorrhage, tarry black, 658.
- „ obstruction, chronic, hard scybalous, 676.
- „ intussusception, red currant jelly, 673.
- „ typhoid fever, pea soup, 63.
- „ yellow atrophy of liver, often clay-coloured, 726.

Strabismus, cerebro-spinal meningitis, 78.

Strain as a factor in valvular disease, 443.

Strangulation of bowel, 672, 673.

Strawberry like tongue in scarlet fever, 166.

Streptococci, in appendicitis, 688.

- „ brain abscess, 893.
- „ bronchitis, acute, 503.
- „ diphtheria, 94, 96.
- „ endocarditis, 433.
- „ erysipelas, 140.
- „ peritonitis, 759.
- „ pleurisy, 599.
- „ pneumonia, 533, 546.
- „ puerperal fever, 144.
- „ renal abscess, 137.
- „ rheumatic fever, 294.
- „ scarlet fever, 163.

Streptococcus conglomeratus in scarlet fever, 163.

Stricture of bile ducts, 745.

- „ intestine, 674, 697.
- „ œsophagus, 625.
- „ trachea and bronchi, 517.

Struma, 392.

Stroke, apoplectic, 867.

Subcutaneous nodules in rheumatic fever, 297.

Subdiaphragmatic abscess, 765.

Subphrenic abscess, 765.

- „ appendicitis, 690.
- „ diaphragm, perforation, 766.
- „ gastric ulcer, 656, 657, 765.
- „ lung abscess, 550, 765.
- „ Röntgen rays, 767.

Subphrenic pyo-pneumothorax, 657, 766.

Substantial emphysema, 520.

Subsultus tendinum in typhoid fever, 66.

Sudamina, in typhoid fever, 65.

Sugar in urine, 330, 792.

Sun stroke, 292.

Suppression of urine, 780 (*see* anuria).

Suppurative appendicitis, 689.

- „ cholangitis, 742.
- „ encephalitis, 893.
- „ gastritis, 649.
- „ hepatitis, 710.
- „ nephritis, 821.
- „ pancreatitis, 750.
- „ peritonitis, 760.
- „ pleurisy, 601.
- „ pyelephlebitis, 709.
- „ tonsillitis, 446.

Suppurative tonsillitis (*see* quinsy).

Suprarenal bodies, diseases, degenerations, 402.

- „ hæmorrhages, 402.
- „ tuberculosis, 402.
- „ tumors, 406.

Suprarenal extract, 401 (*see* adrenalin).

Surgical kidney, 821.

Sweating, in malaria, 243.

- „ phthisis, 579.
- „ rheumatism, acute, 296.
- „ typhoid fever, 62.

Sydenham's chorea, 965.

Symmetrical gangrene, 996.

Sympathetic nerves and ganglia in Addison's disease, 403.

Symptomatic parotitis, 615.

Syncope, fatal, in cardiac disease, 418, 419, 453.

- „ paracentesis thoracis, 606.

Syncope, local, 997.

Syphilis, 182.

- „ adenoids, 495.
- „ albuminuria, 788.
- „ alopecia, 185.

Syphilis—

- „ anæmia, 187.
- „ aneurism, 188, 429.
- „ angina pectoris, 468.
- „ animals, lower, 183.
- „ aortic incompetence, 451.
- „ arteritis, 475, 476.
- „ arterio-sclerosis, 188.
- „ atheroma, 478.
- „ bacillus of Lustgarten, 183.
- „ bile duct occlusion, 34, 741, 745.
- „ chancre, hard, 184, 185.
- „ Colles' law, 189.
- „ condylomata, 186, 697.
- „ degeneration, waxy, 30, 188, 678.
- „ duration of second stage, 185.
- „ endarteritis, 185, 188, 476.
- „ endocarditis, chronic, 443.
- „ eruptions, 186, 188.
- „ facies, hereditary, 189.
- „ fever, 186.
- „ general paralysis of insane, 188, 945.
- „ gummata, 185, 187, 590, 697, 719.
- „ heredity, 189.
- „ Hutchinson's teeth, 189.
- „ hypodermic injections, 191.
- „ incubation periods, 185, 186.
- „ icterus neonatorum, 34, 741, 745.
- „ infection methods, 182.
- „ „ strength, 184.
- „ interstitial, inflammation, 188.
- „ intestine, 697.
- „ iritis, 186, 189.
- „ keratitis, 189.
- „ kidneys, 773, 799, 800, 806, 818.
- „ larynx, 500.
- „ leprosy transmission, resemblance, 124.
- „ lesions, 184, 185.
- „ liver, 189, 714, 719.
- „ lymphatic glands, 184.
- „ marriage, 184.
- „ mercury, 190.
- „ miscarriages, 190.
- „ mucous patches, 186.
- „ myocarditis, 423.
- „ nephritis, 799, 806, 811, 818.
- „ ostitis, 188, 189.

Syphilis—

- „ periarteritis, nodosa, 188, 478.
- „ periostitis, 187, 188.
- „ pharyngitis, 619.
- „ pneumonia, white, 590.
- „ potassium iodide, 191, 192.
- „ retinitis, 185.
- „ snuffles, 189.
- „ spleen, 189.
- „ spironaema pallidum, 183.
- „ stomatitis, 614.
- „ stricture of intestine, 674.
- „ œsophagus, 188.
- „ rectum, 188.
- „ tabes dorsalis, 188, 929.
- „ treatment, duration, 190.
- „ ulcers, superficial, 185.
- „ „ deep, 187.

Syphilitic cirrhosis, liver, 718.

Syringomyelia, 862.

- „ amyotrophic lateral sclerosis, 863.
- „ Babinski's sign, positive, 863.
- „ bedsores, 864.
- „ bone atrophy, 864.
- „ gliosis, 860.
- „ leprosy, 864.
- „ muscular atrophy, 861.
- „ nails, brittle, 864.
- „ pain, sense lost, 863.
- „ pachymeningitis cervicalis, 847, 864.
- „ skin, glossy, 864.
- „ sensation, ordinary, 864.
- „ whitlows, 864.

Tabes dorsalis, 929.

- „ age, 929.
- „ alocheiria, 933.
- „ anæsthesia, 933.
- „ arthritis, 931, 935.
- „ ataxia, 932.
- „ crisis, 935.
- „ eye symptoms, 934, 935.
- „ „ Argyll Robertson pupil, 934.
- „ „ diplopia, 935.
- „ „ optic atrophy, 931, 934.
- „ „ ptosis, 935.
- „ „ spinal myosis, 934.
- „ general paralysis of insane, 945.
- „ knee-jerks, 933.
- „ lightning pains, 932.

Tabes dorsalis—

- „ muscular sense, 933.
- „ paralysis, 932.
- „ pre-ataxic stage, 932.
- „ perforating ulcer, 935.
- „ Romberg's sign, 933.
- „ syphilis, 929.

Tache cérébrale, significance, 853.

Tachycardia, 464.

- „ causes, 465.
- „ paroxysmal, 465.

Tæniæ, varieties, 255.

Tapeworms, 255.

Tapping in ascites, 757.

- „ hepatic cirrhosis, 722.

Taste, affections of the sense, 1047.

- „ loss, 1048.
- „ metallic, 1048.
- „ perversion, 1048.
- „ smell, 1047, 1048.
- „ test, 1048.

Tattooing, 35.

Tea, cardiac palpitation caused by, 463.

Teachers' node, 499.

Teeth, actinomycosis, 129.

- „ fetid stomatitis, 611.
- „ Hutchinson's, 189.
- „ pyorrhœa alveolaris, 611.
- „ rickets, 315.

Telegraphists' cramp, 978.

Temperature, inverse type in acute tuberculosis, 563.

Temperature sense, lost in syringomyelia, 863.

Temporal lobe, tumors, 954.

Temporo-sphenoidal lobe, auditory word centre, 961.

Tendon reflex, 840 (*see* reflexes).

Tendon transplantation in hemiplegia, 879.

Tenesmus in proctitis, 680.

Tenth nerve, 1059.

Tertian ague, 238.

Tetanus, 103.

- „ anterior horn cells, 944.
- „ antitoxin, 109.
- „ asphyxia, 107.
- „ bacillus, 103.
- „ cephalic, 107.
- „ cardiac failure, 107.
- „ exhaustion, 107.
- „ fever, 107.
- „ idiopathic, 104.
- „ mortality, 108.
- „ neonatorum, 106.
- „ opisthotonos, 107.

Tetanus—

- „ pain, 107.
- „ pyogenic germs, 105.
- „ risus sardonicus, 107.
- „ spasms, early, 106.
- „ „ tonic, 107.
- „ „ paroxysmal, 107.
- „ strychnine poisoning, contrast, 108.
- „ toxin affinity for nervous tissues, 105.
- „ wound, dirty, 104.
- „ absence, 107.

Tetany, 980.

- „ Chvostek's symptom, 981.
- „ gastric and intestinal disorders, 980.
- „ gastric dilatation, 651.
- „ posterior gastro-enterostomy, 981.
- „ after infections, 980.
- „ rickets, 317, 980, 982.
- „ after thyroidectomy, 980.
- „ toxic, 980.

Therapy serum, diphtheria, 101.

- „ plague, 218.
- „ tetanus, 109.

Third nerve, 1036.

- „ paralysis, 1037.

Thirst in diabetes, 333, 339.

Thomsen's disease, 1069.

Thoracic duct, rupture, 596.

- „ tubercle bacilli, travelling, 562.

Thorax, deformity, chronic pericarditis, 416.

- „ emphysema, 522.
- „ pleurisy, 602.
- „ rickets, 315.
- „ chronic tonsillitis, 623.

Thread worms, 261.

Thready pulse in peritonitis, 763.

Thrill, diastolic, 452.

- „ presystolic, 450.

Thrombi, varieties, 7.

- „ white, left auricle, 449.

Thrombosis, 7.

- „ cerebral arteries, 884.
- „ cerebral sinuses, 889.
- „ chlorosis, 889.
- „ marantic, 889.
- „ meningitis, 889.

Thrush, 610.

Thymic asthma, 407.

Thymus gland, 406.

- „ Addison's disease, 397.
- „ acromegaly, 1001.

Thymus gland—

- „ asthma, 407.
- „ dyspnœa, 407.
- „ exophthalmic goitre, 407.
- „ hypertrophy, 407.
- „ leukæmia, 397.
- „ lymphadenoma, 397.
- „ suffocation, 407.
- „ sudden death, 407.

Thyroid extract, administration, 392.

Thyroid gland, 390.

- „ acromegaly, 1001.
- „ accessory, 391.
- „ cretinism, 395.
- „ enlargement, goitre, 392.
- „ exophthalmic goitre, 397.
- „ inflammation, 392.
- „ myxœdema, 394.
- „ parathyroid glands, 391.
- „ tumors, 400.

Thyroidectomy, 391, 393, 397, 400.

- „ tetany, 981, 982.

Thyroidine, 350.

Tic, impulsive, 970.

Tic douloureux, 1005.

- „ empyema of antrum, 1005.
- „ extirpation of Gasserian ganglion, 1010.

Tinnitus aurium, 1055.

Tobacco poisoning, 286.

- „ cardiac depression, 286.
- „ chronic gastritis, 642.
- „ insomnia, 286.
- „ chronic laryngitis, 498.
- „ pharyngitis, chronic, 617.

Tongue, biting, in epilepsy, 972.

- „ eczema, 609.
- „ geographical, 609.
- „ jerking, chorea, 966.
- „ paralysis of, bulbar paralysis, 919.
- „ strawberry, scarlet fever, 166.
- „ tremor, alcoholism, 275.
- „ „ bulbar paralysis, 919.
- „ „ general paralysis, 946.
- „ typhoid fever, 63, 64, 74.
- „ woody, actinomycosis of cattle, 130.

Tonsillitis, 616.

- „ acute, 619.
- „ chronic, 622.
- „ follicular, 619.
- „ rheumatism, 435, 617, 622.
- „ suppurative, 623 (see quinsy).

Tonsils, hypertrophy, 622.

- „ inflammations, 619.

Tonsils, hypertrophy, deafness, 623.

- „ „ mental dulness, 623.
- „ „ mouth breathing, 623.
- „ „ nasal speech, 623.
- „ „ pigeon breast, 623.

Tophi, 321.

Torticollis, 1064.

- „ congenital, 1064.
- „ spasmodic, 1065.

Tormina, dysentery, 209.

Toxic gastritis, 648.

Trachea, obstruction, dyspnœa, 517.

- „ „ partial, 517.
- „ „ total, 517.
- „ „ sudden death, 517.

Tracheotomy, in diphtheria, 102.

- „ œdema glottidis, 498.

Trance, in hysteria, 989.

Transfer of pain in hysteria by "suggestion," 987.

Traube's plugs in bronchiectasis, 513.

Traumatic neuroses, 591.

Trematodes, 254.

Tremor, in alcoholism, 275.

- „ exophthalmic goitre, 398.
- „ intention of disseminated sclerosis, 950.
- „ general paralysis, 946.
- „ paralysis agitans, 963.
- „ senile, 963.
- „ sleeping sickness, 252, 253.

Trichina spiralis, 266.

- „ adult, 266.
- „ anæmia, 267.
- „ diarrhœa, 267.
- „ delirium, 267.
- „ fever, 267.
- „ eosinophilia, 267.
- „ larva, 266.
- „ œdema, 267.
- „ sweating, 267.
- „ tremors, 267.
- „ typhoid fever, resemblance, 267.

Trichiniasis, 266.

Trichocephalus dispar, 263.

- „ anæmia, 264.

Tricuspid regurgitation, 456.

- „ murmur tricuspid systolic, 456.
- „ „ tricuspid presystolic, 457.
- „ stenosis, 456.

Tricuspid valve, incompetence, 456.

- „ stenosis, 456.

Trigeminal neuralgia, 1005.
 „ nerve, 1043.
 Trismus, 1047.
 „ hysteria, 1047.
 „ tetanus, 107, 1047.
 Trommer's test for sugar, 331.
 Trophic diseases, 995.
 Tropical dysentery, 209.
 „ abscess of liver, 711.
 Trousseau's symptom in tetany, 981.
 Trypanosoma, nagana, 251.
 „ sleeping sickness, 250.
 Tube casts, 793.
 „ blood cell, 777, 795.
 „ cellular, 795.
 „ epithelial, 776, 795.
 „ fatty, 776, 795.
 „ granular, 777, 795.
 „ hyaline, 776, 777, 794.
 „ significance, 794, 795, 804.
 „ waxy, 776, 794.
 Tubercle bacillus, 112.
 Tubercula dolorosa, 1020.
 Tuberculin, 122, 580.
 Tuberculosis, 109.
 „ abscess, 118, 119.
 „ acid-fast bacilli, 112.
 „ Addison's disease, 403.
 „ ærogenic, 564.
 „ age influence, 110.
 „ albuminuria in pulmonary, 569.
 „ alcoholism, chronic, 110.
 „ anæmia, 119, 578.
 „ animals, 113.
 „ arteritis, chronic, 476.
 „ bacillus, 109.
 „ „ caseous material, 567, 568.
 „ „ destruction, 582.
 „ „ significance, discharges, 120.
 „ „ sputum, 115, 653, 569, 577.
 „ blood spread, 116, 561, 562.
 „ bronchitis, acute, 561, 565, 567.
 „ bronchogenic, 564.
 „ cavity formation in pulmonary, 566, 573.
 „ caseation, 114, 562, 565, 568, 572.
 „ cells, 113.
 „ choroid, 563.
 „ constructive changes, 113.
 „ cure, spontaneous, 122, 572.

Tuberculosis—
 „ crusade, modern, against, 120, 573.
 „ destructive changes, 114.
 „ diabetes mellitus, 110, 335.
 „ diarrhœa, 576.
 „ diminishing incidence, 121.
 „ dust, 110.
 „ dyspnœa, pulmonary forms, 563, 569, 578.
 „ endocarditis, 435.
 „ environment, 111, 583.
 „ evolution of pulmonary lesions, 567.
 „ fever, 119, 563, 569, 578.
 „ fibrous tissue, 115, 571.
 „ hæmoptysis, 529, 531, 569, 573, 577.
 „ heart, 576.
 „ heredity, 110, 111.
 „ infection, methods, 112, 561, 562.
 „ „ mixed, 119, 569, 574.
 „ „ pneumococcus, 569.
 „ insomnia, in pulmonary, 569.
 „ intestinal, 575, 674, 694.
 „ kidney, 576, 818.
 „ Koch's views, 113, 121.
 „ laryngeal, 499, 500, 575.
 „ leprosy, 127.
 „ leucocytosis, absence, 120.
 „ liver, 576.
 „ London Congress, 113.
 „ lymphatic glands, 115.
 „ lymph spread, 115.
 „ marriage, 121.
 „ meningitic, 848, 852.
 „ mesenteric glands, 116.
 „ miliary, acute, 114, 561.
 „ „ chronic, 562.
 „ milk, 113.
 „ myocarditic, 423.
 „ night sweats, pulmonary, 579.
 „ occupation, 110, 583.
 „ opsonic treatment, 587.
 „ pericarditic, 411, 416, 576.
 „ peritoneum, pleuritic, 599.
 „ pleural changes, pulmonary, 565, 574.
 „ pneumonia, interstitial, parenchymatous, 562, 568, 571.
 „ pneumothorax, 574.
 „ pneumoconioses, 556.
 „ predisposing causes, 109, 110.
 „ primary focus, 117, 567.

Tuberculosis—

- „ pulmonary, 111, 117, 546, 561.
- „ pyogenic germs, 118, 120, 574.
- „ quiescence, 115, 573, 581.
- „ sclerosis, cord, 944.
- „ serous membranes, 576.
- „ smegma group, bacilli, 112.
- „ spleen, 564, 576.
- „ sweating, 564, 579.
- „ toxin, 114, 122.
- „ typhoid-like symptoms, 563, 569.
- „ tuberculin, 122.
- „ valvular disease, heart, 576.
- „ vulnerability of tissue, 111.
- „ wasting, 120, 578.

Tubular diarrhoea, 684.

Tuffnell's starvation treatment of aneurism, 485.

Tumors, brain, 951.

- „ cord, 951.

Twist, kink of bowel, 674.

Tympanites, intestinal obstruction, 675.

- „ peritonitis, 762.
- „ typhoid fever, 65, 72.

Typhoid fever, 58.

- „ abortive, 67.
- „ albuminuria, 788.
- „ ambulatory, 67.
- „ anæmia, 61, 64.
- „ agglutination test, 68.
- „ arteritis, 476.
- „ bacillus, 59.
- „ bacteriological examination, 68.
- „ baths, cold, 69, 70.
- „ bedsores, 66, 72.
- „ brain paralysis, 69.
- „ bronchitis, 66, 74, 505, 558.
- „ cardiac failure, 66, 72.
- „ carriers of bacillus, 59.
- „ children, 73.
- „ coma vigil, 64.
- „ constipation, 63.
- „ decubitus dorsal, 64, 65.
- „ delirium, 61, 64, 65, 73, 74.
- „ diarrhoea, 63, 71, 75.
- „ diet, 70.
- „ dust, 59.
- „ emphysema, 66.
- „ epistaxis, 61.
- „ erysipelas, 66.
- „ fever, 62, 65, 67, 74.
- „ flies, 59.

Typhoid fever—

- „ hæmorrhage, intestine, 62, 66, 67, 71.
- „ „ nose, 61.
- „ hyperpyrexia, 47, 48, 69, 73.
- „ immunity, 58.
- „ insomnia, 72.
- „ laryngeal ulceration, 66.
- „ Leibermeyer's treatment, 69, 70.
- „ leucocytosis, absence, 60, 68.
- „ lysis, 62.
- „ meningitis, 61, 852.
- „ mesenteric glands, 61, 65.
- „ meteorism, 65, 72.
- „ milk, 59.
- „ nephritis, 799.
- „ oysters, 59.
- „ parotitis, symptomatic, 66, 615.
- „ perforation, intestinal, 66, 67.
- „ peritonitis, 67, 72.
- „ Peyer's patches, 59, 60, 63.
- „ pleurisy, 66, 599.
- „ pneumonia, 61, 65, 546, 558.
- „ ptomaine poisoning, resemblance, 259.
- „ pulse, 61.
- „ rash, 64, 66, 74.
- „ relapses, 64.
- „ seasonal frequency, 59.
- „ simple continued fever, 144.
- „ solitary glands, 59, 60, 63.
- „ spleen, 60, 63.
- „ stupor, 62, 64.
- „ stools, 63, 64, 74.
- „ stricture, intestinal, 674.
- „ subsultus tendinum, 66.
- „ sudamina, 65.
- „ tongue, 63, 64, 74.
- „ ulceration, intestinal, 60, 66.
- „ „ laryngeal, 66.
- „ „ pharyngeal, 619.
- „ urine, 63, 65.
- „ Widal test, 68.

Typhoid state, pyæmia, 138.

Typhoid type, malignant endocarditis, 440.

Typho-malarial fever, 244.

Typho-remittent fever, 244.

Typhus fever, 192.

- „ abortion, absence, 197.
- „ age, 193.
- „ albuminuria, 197.
- „ alcohol, 200.

Typhus fever—

- „ armies, 194.
- „ asthenia, 198.
- „ bedsores, 197.
- „ bronchitis, 197.
- „ coma, 197, 198.
- „ convulsions, 197.
- „ delirium, 196, 197.
- „ facies, 194, 196.
- „ fever, 194, 196.
- „ gangrene, 197.
- „ geographical distribution, 193.
- „ jaundice, 741.
- „ muscular debility, 194, 196.
- „ necrosis of cardiac muscle, 198.
- „ pneumonia; 197.
- „ pupils contracted, 197.
- „ rash, hæmorrhagic, 195.
- „ „ mulberry, 195.
- „ „ preliminary, 194.
- „ smell, bodily, 197.
- „ subsultus tendinum, 196.
- „ typhomania, 196.
- „ urine, 196.

Tyrosin, liver, yellow atrophy, 725.
 „ urine, 726, 787.

Ulcer, duodenum, 655, 660.

- „ colon, 684.
- „ dysenteric, 208.
- „ foot in tabes dorsalis, 935.
- „ œsophagus, 627, 655.
- „ mouth, 611.
- „ stomach, 653.
- „ peptic, 653.
- „ malignant, 663.
- „ typhoid and tubercular compared, 60.

Ulcerative colitis, 684.

- „ relation to dysentery, 684.

Ulcerative endocarditis, 438.

Ulnar nerve paralysis, 1025.

Umbilical cord hæmorrhage, 741.

Undulant fever, 87.

Uræmia, 782.

- „ Cheyne-Stokes breathing, 783.
- „ coma, 782.
- „ consciousness, 780, 783.
- „ delirium, 782.
- „ diarrhœa, 783.
- „ dyspnœa, 783.
- „ epileptiform convulsions (Jacksonian), 783.
- „ hemiplegia, 783.
- „ insomnia, 783.

Uræmia—

- „ latent, 780.
- „ nephritis, 804, 808, 814-817.
- „ urea, excretion, 781.
- „ vomiting, 783.

Urates, urine, 784.

Uratric deposits, gout, 321.

Uric acid calculus, 796.

- „ cayenne pepper-like deposit, 784.
- „ gout, 318.
- „ reaction in leukæmia, 783.
- „ showers, 319.
- „ urine, 784.

Uric acid diathesis, 784.

Urinary calculi, 795.

- „ composition, 796.
- „ cyst, 796.
- „ gravel, 797.
- „ hydronephrosis, 822.
- „ oxalate, 796.
- „ phosphatic, 796.
- „ position, 797.
- „ pyelitis, 820.
- „ results, 797.
- „ uric acid, 796.

Urine disturbances, secretion, 778.

Urine, acetone, 333, 792.

- „ albumin, 787.
- „ albumose, 789.
- „ amount, 778.
- „ bile, 793.
- „ blood, 790.
- „ chlorides, 330, 785.
- „ cholera, 83, 779.
- „ chyle, 790.
- „ cystin, 787.
- „ diacetic acid, 793.
- „ diphtheria, 98.
- „ fat, 746, 790.
- „ febricula, 145.
- „ glycuronic acid, 792.
- „ gout, 319.
- „ hæmoglobin, 247, 388, 389, 791.
- „ indican, 746, 786, 793.
- „ kreatinin, amount, 784.
- „ leucin and tyrosin, 725, 726, 787.
- „ mucin, 792.
- „ oxalates, 785.
- „ phosphates, 785.
- „ pigments, 785, 792.
- „ pneumonia, 537, 538.
- „ pus, 790.
- „ small-pox, 151.
- „ specific gravity, 780.

Urine—

- „ sulphates, 786.
- „ sugar, 330, 792.
- „ suppression, 780.
- „ tubercle bacillus, 800, 819.
- „ tube casts, 793.
- „ typhoid bacillus, 68, 800.
- „ typhoid fever, 63, 65.
- „ typhus fever, 196.
- „ urea, amount, 330, 538, 781.
- „ uric acid, amount, 784.
- „ yellow fever, 203.

Urobilin, 786.

Uroerythrin, 786.

Urino-genital tuberculosis, 818.

Vaccinia, 159.

Vaccination, 160.

- „ age, best for, 161.
- „ eruption, 161.
- „ Jenner, 159.
- „ re-vaccination, 161.

Vaccine lymph, 159.

- „ effect of addition of glycerine, 159.

Vaccine treatment, cholera, 83.

- „ plague, 218.

Vagabonds discoloration of skin, 405.

Valvular disease of heart, 443.

Varicella, 162.

Variola, 149 (*see* small-pox).

Varioloid, 157.

Vasomotor diseases, 995.

Vegetations on heart valves, 434, 435, 436, 438.

Veins, cerebral thrombosis, 889.

Veins, cesophageal hæmorrhage from hepatic cirrhosis, 720.

Venesection (*see* blood-letting).

Ventricles of brain, dilatation of, hydrocephalus, 859.

- „ hæmorrhage, 880.
- „ tapping, 860.

Vertebra, caries, 617, 899.

Vertigo, 1057.

- „ auditory, 1057.
- „ brain tumor, 953.
- „ cardiac, 1057.
- „ gastric, 645, 1057.
- „ hysterical, 1057.
- „ ocular, 1057.

Vestibular nerve, lesions, 1056.

Ventricular hæmorrhage, 528.

Virus fixé, 232.

Vision, double, fourth nerve paralysis, 1039.

Vision—

- „ ocular spasm or paralysis, 1057.
- „ sixth nerve paralysis, 1040.
- „ tabes dorsalis, 935.

Vocal resonance, chronic phthisis, 579.

- „ pneumothorax, 597.

Voice of "dead" quality in adenoids, 495.

Volvulus, 674.

Vomit, black, yellow fever, 202, 203.

- „ "coffee ground," gastric cancer, 665; ulcer, 657.

Vomiting, Addison's disease, 404.

- „ brain abscess, 895.
- „ „ tumor, 952.
- „ enteritis, 685.
- „ gastric cancer, 665.
- „ „ catarrh, 639, 644.
- „ „ crises, 935.
- „ „ ulcer, 657.
- „ fecal, 792.
- „ hepatic cirrhosis, 720.
- „ yellow atrophy, 726.
- „ meningitis, 852.
- „ nephritis, acute, 804.
- „ „ chronic, 816.
- „ peritonitis, 762.
- „ phthisis, chronic, 578.
- „ pneumonia, 539.
- „ uræmia, 783.
- „ whooping-cough, 179, 180.
- „ yellow fever, 202.

Wall paper, poisoning by arsenic, 288.

Warnings, impending apoplexy, 871.

Wart pox, 157.

Washing out stomach, 652.

Waterbrash, 644.

Waterhammer pulse, 453.

Water, infection, in cholera, 81.

- „ typhoid fever, 59.

Weil's disease, 741.

Werlhof's disease, 383.

Wernicke's pupil reaction, 1035, 1038.

"Wet pack," 48.

Whip worm, 263.

White infarct in kidney cortex, 12.

White softening in brain, 12.

White thrombi in left auricular appendix, 449.

Whooping-cough, 178.

- „ age, 178.
- „ bromoform, 180.
- „ bronchitis, 179, 505.

Whooping-cough—
 „ convulsions, 179, 977.
 „ convulsive stage, 179.
 „ dyspnœa, 178.
 „ epidemicity, 178.
 „ epistaxis, 179.
 „ fever, 178.
 „ glycosuria, temporary, 179.
 „ hydrocyanic acid, 180.
 „ immunity, 178.
 „ measles, 178.
 „ mortality, 180.
 „ pneumonia, 179, 545.
 „ pulmonary collapse and emphysema, 179, 524.
 „ tongue, ulceration under, 179.
 „ vomiting, 179, 180.
 Woolsorters' disease, 222, 223, 224.
 Word blindness, 961.
 „ deafness, 961.
 Wormian bones in hydrocephalus, 860.
 Worms, diseases caused by, 254.
 Wrist drop in alcoholic multiple neuritis, 1016.
 „ lead paralysis, 1017.
 „ writer's cramp, 979.
 Writing, faulty method, 978.
 Wryneck, 1064.
 „ congenital, 1064.

Wryneck—
 „ spasmodic, 1065.
 Xanthelasma, 35.
 Xerostomia, 615.
 Yellow fever, 201.
 „ albuminuria, 203, 204.
 „ anuria, 203, 780.
 „ bacillus ictteroides, 202.
 „ „ minute, 202.
 „ coma, 203.
 „ convulsions, 203.
 „ delirium, 203.
 „ fever, 203.
 „ glomerulo-nephritis, 202.
 „ hematemesis (black vomit), 202, 203.
 „ immunity, 201.
 „ jaundice, 203, 741.
 „ mosquitoes, 202.
 „ urea in blood increased, 202.
 „ urine, 203.
 Yellow tubercle, 114.
 „ softening in cerebral embolism, 885.
 Yellow vision, 739, 741.
 Yersin's serum, in plague, 218.
 Zigzag lines, light in migraine, 983.
 Zona, 938.
 Zoster, herpes, 938.

